THE HEART IN EARLY LIFE

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THE HEART IN EARLY LIFE

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DEDICATED TO

JAMES MACKENZIE

M.D., LL.D., F.R.C.P.

WITH SINCERE RESPECT AND GRATITUDE

PREFACE

When I go back to my student days and try to remember the teaching on the subject of the heart in early life, my recollection is that it was almost entirely ignored. In those days we were left to find out for ourselves what differences, if any, existed between the conditions present in young hearts and in old. The text-books did not throw much light on the subject. In the course of professional work the differences forced themselves on one's notice, and my own experience of young hearts had to be gained slowly and somewhat painfully.

This book is an attempt to enable the young practitioner to fill up some blanks which may have been left after his medical course has been finished. It is not a systematic text-book, and it has no relation to examination work. It deals with the clinical problems of cardiac disturbances and diseases during childhood and youth, as they present themselves in the ordinary routine of practice. I have tried to take advantage of all the more recent teaching on cardiology, and of instrumental methods in the determination of facts and the elucidation of problems. More especially have I directed attention to certain pitfalls which seem to beset us all in connexion with the subject, and which may be summed up as a tendency to treat too seriously the functional disturbances, and to treat too lightly disease of the heart in its earlier stages.

The standard works bearing on diseases of children have been consulted with much benefit, but their method of teaching the subject has not been adopted, for reasons which will be found in the text. I desire to acknowledge with gratitude the stimulus which I have received from the writings and teaching of Dr. James Mackenzie. The additions made to the science of cardiology by that gifted physician are so numerous and so important that all writers on the subject borrow, consciously or unconsciously, from him. I have borrowed freely, at times consciously and with acknowledgement, and at times probably unconsciously and without acknowledgement. But if a reader of these pages finds anything which strikes him as new and true and in accordance with his own experience he may safely ascribe it to Dr. Mackenzie's teaching. The pathology of the subject has been but lightly touched on, but the work of Dr. Poynton and Dr. Carey Coombs has been none the less appreciated. I have also found much help in estimating the value of the information to be obtained by means of the electro-cardiograph in the writings of Dr. Thomas Lewis and Dr. W. T. Ritchie. For many of the tracings which are used as illustrations, and for much help generally, I am greatly indebted to my late house-physicians, Dr. W. P. Murray, Dr. R. G. Gordon, Dr. Hugh Paterson, and Dr. S. H. Hay.

Wimpole Street, W.
May 1914.

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LIST OF ILLUSTRATIONS

INTERPRETATION OF POLYGRAPH TRACINGS

The majority of the illustrations which follow are from tracings taken with Mackenzie's ink polygraph. They are for the most part simple and easily followed. The following explanation may help those who are not familiar with the instrument.

The notched line at the top of each tracing is made by the time marker, and the space between two notches represents one-fifth of a second. Thirty of the spaces will therefore correspond to six seconds, and three hundred to one minute.

The lower tracing is taken from the radial or the brachial artery. In children the brachial artery at the bend of the elbow is more suitable than the radial for an arterial tracing, because it is a larger vessel, and because the button spring can be fixed in position more easily at the elbow than at the wrist. The characters of the pulse tracing thus obtained are sufficiently well known. It is quite easy in the case of children to get a pulse tracing of considerable amplitude, but this must not be interpreted as representing a powerful cardiac action. The amplitude of the pulse tracing is largely dependent on the exact adjustment of the button spring over a superficial vessel, and the amount of pressure made by the spring. In other words, the result is instrumental. At the same time a hypertrophied heart acting slowly will give more

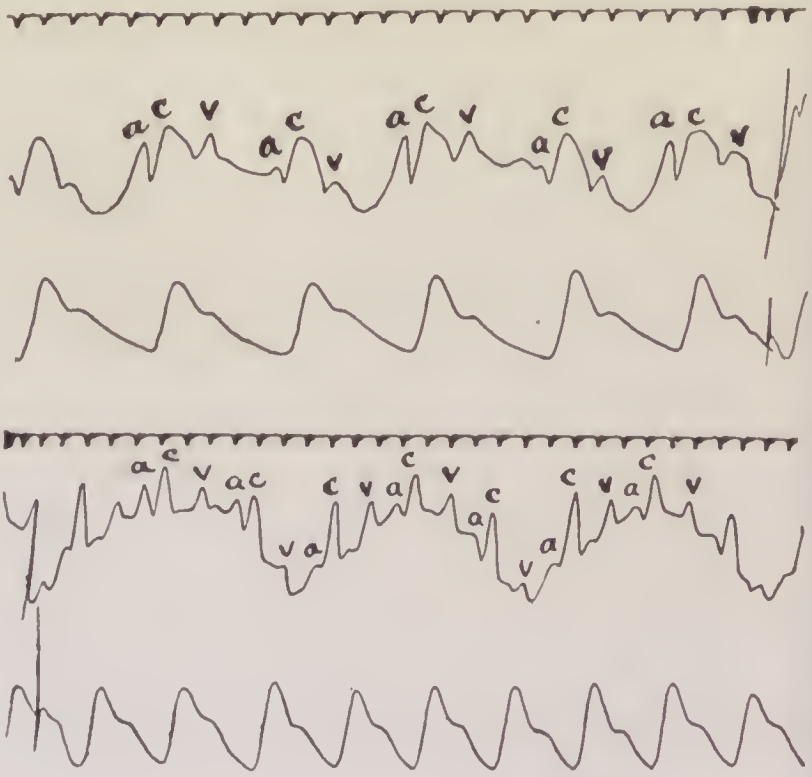
readily a large amplitude of pulse tracing than a weak heart acting quickly. The exact rate of the pulse can be accurately measured from the radial tracing. If thirty of the notched spaces in the upper line are measured with compasses, and the number of beats in the radial tracing occupying the same space are counted, we obtain the number of beats in six seconds. If this number is multiplied by ten we get the number of beats (cardiac pulsations) per minute. The appearance of the radial tracing is affected by the rate at which the tracing-paper moves. When a quick succession of beats is desired, as for the purpose of detecting the presence of any irregularity, the tracing-paper is made to move slowly. The notched line at the top will then show very short intervals between the notches. When a more extended radial tracing is desired, as for the purpose of analysis or for comparison with an accompanying venous tracing, the tracing-paper is made to move quickly. The notched line at the top will then show much longer intervals between the notches.

The upper tracing in many of the illustrations is the so-called venous pulse, which registers more especially the movements of the right auricle. It is taken from the jugular bulb on the right side of the neck. A series of waves—known as *a*, *c*, and *v*—is recorded, and these waves have to be identified by measurements taken from the radial tracing. 'We find in the neck tracing a small wave due to the impact of the carotid pulse—the *c* wave—and we recognize it by its constant relationship in

time to the radial. As the wave that travels down the arm to the radial takes some time, we find that the radial pulse appears generally one-tenth of a second after the carotid. When, therefore, we want to find the carotid wave in the neck tracing, we seek a wave one-tenth of a second before the radial pulse' (Mackenzie). One-tenth of a second corresponds to one-half of a space in the notched line at the top of the tracing. The wave preceding the carotid wave is produced by the contraction of the auricle, and is known as the *a* wave. It occurs under normal conditions one-fifth of a second before the carotid wave. There is a third wave, called the *v* wave, which occurs after *c*, and is due to the storing up of blood in the auricle during the ventricular systole. In time this wave is found to correspond with the dicrotic notch in the radial tracing, and on measurement the apex of the *v* wave is found to correspond exactly with the bottom of the dicrotic notch. In measuring out the venous and radial tracings it is necessary to have ordinates at intervals in the course of a long tracing. For this purpose the revolving cylinder is stopped, and the tracing pens are drawn vertically across the venous and radial tracings. From these ordinates measurements are made as described above.

The accompanying illustrations represent a jugulo-brachial tracing of normal character. The lower part of each tracing is from the brachial artery. The upper part is from the venous pulse in the neck. The various waves—*a*, *c*, and *v*—in the latter have been differentiated by measurement with the

brachial tracing and are indicated by letters. The first tracing is taken with the cylinder revolving more quickly, and the second with it revolving more slowly. The ordinates for measurement are shown. The large undulations in the venous tracing



are due to the movements of respiration, which are commonly exaggerated in childhood. In order to make the venous tracing pursue a steady course, like the brachial, it is necessary for the patient to hold his breath or to breathe very quietly. When actual dyspnœa is present the taking of a venous tracing may be very difficult or impossible.

CHAPTER I

GENERAL INTRODUCTION

Recent Views and Methods

The great progress made within recent years in connexion with the study of the functions and diseases of the heart has led a good many of us to revise our former ideas on these subjects. Not only have many new facts been elicited, but many old views have been combated. It may be added that the new facts are becoming day by day more firmly established by the experience of those who have tested them, and that the foundations of many of the old beliefs seem to be crumbling away because of their unscientific structure.

In some ways it is to be regretted that the student makes the acquaintance of heart disease in the wards of a hospital, because the cases submitted to him are almost entirely those illustrating the later stages of disease. As he masters the mysteries of murmurs and dilatation and irregularities, he unconsciously associates these in the mental picture with hearts broken down from advanced degeneration, rheumatic, alcoholic or arterio-sclerotic in origin. The result is, that when he qualifies he goes forth to practise with the idea that a murmur or an irregularity in the cardiac region implies the presence of

heart disease, and that of a serious nature. I do not think that, as a rule, the student learns anything about the numerous forms of cardiac disturbance of a non-organic kind which he will have to deal with in practice.

If one considers the natural history of heart disease it would seem to be a better plan for a student to commence his study of the subject in more youthful patients. It is in early life that the heart is attacked by the most common cause of organic disease, namely, rheumatic infection. It is at this period that one can trace the various signs and symptoms as they progress. One can see in a much less complicated manner the results of endocardial and myocardial and pericardial infection. One can observe the results that follow from one attack and from repeated attacks. One can learn the fact, not often in evidence in adult life, that an infected heart may make a complete recovery. The student would also become acquainted with a large number of functional disturbances and disorders, and would learn how to distinguish between these and organic diseases. With a knowledge of heart affections in childhood and youth the student would then progress in a natural manner to a study of those diseases in adult life and old age.

The term 'heart disease' is one of dread significance to the general public, and even the alternative expression of 'something wrong with the heart', frequently used by medical men in conveying information to relatives, is not calculated to soothe

anxious minds. There is a reasonable basis for this dread of the term 'heart disease', because sudden and unsuspected death does often occur in the adult subjects of cardiac trouble. The medical student learns as facts that heart disease is a common cause of death in adult life, and that certain signs and symptoms connected with the heart are of grave significance. He next assumes that the same significance attaches to the signs and symptoms of heart disease in early as in later life. He goes forth to practise, and his prognosis and treatment of cardiac disorder and disease in children and in youths will be based on his knowledge of the adult types of disease, irrespective of any difference in the real significance of the signs and symptoms at different ages. The assumption is wrong and the result is unfortunate. All of us tend to fall into this error unless we observe and consider the facts of heart disease in early life with an open mind, and irrespective of any views based on the study of adult cases.

Cardiac disease and cardiac disturbance may be caused by various factors in adult life which are not present in childhood, and vice versa. In adult life we have such factors to deal with as degenerated arteries (arterio-sclerosis, atheroma), as chronic bronchitis and emphysema, as chronic renal disease, and as various poisons in the system, e.g. alcohol, syphilis, &c. These are of immense importance in determining the power of the heart in maintaining an efficient circulation, while in childhood they are for all practical purposes simply non-existent and

do not enter at all into the problems connected with heart efficiency. On the other hand, the period of childhood and youth has its own peculiarities and problems. We have to note an instability about the cardiac action which is not present in the fully-developed adult heart. This instability would appear to be both in the nervous mechanism—central and peripheral—which commands the heart, and in the heart itself. The cardiac action is much more easily disturbed because the cardiac apparatus is not yet fully developed. Hence comes a large number of what may be called developmental disorders and disturbances. As contrasted with the raised arterial pressure of advancing years, we find in early life a tendency to a lowered vasomotor tone, and more especially to an unstable vasomotor tone in the arteries. This reacts on the heart and may lead to various symptoms, often described as cardiac, but more properly referred to the vascular tonicity. In childhood the heart has not only to maintain an efficient circulation already established, but it has to adapt itself to the changing circulation brought about by the growth and development of the body as a whole. A satisfactory correlation between the needs of the body and the efficiency of the circulation can only be brought about by a steady growth and development in the cardiac structures. Temporary disturbances in what have usually been regarded as the physiological limitations of this correlation may seem to be present, but must not be hastily taken as evidences of heart disease or

heart weakness. In dealing with the heart in early life we must allow a considerable range in its physiological manifestations, without calling them pathological, just as we allow a considerable range to the manifestations of cerebral activity, without calling them pathological, as compared with those of adult life. With the completion of the period of growth and development the greater stability and the orderly mechanism of the adult heart will be established.

This factor of the steady growth and development of the heart must also be considered in relation to organic cardiac disease from rheumatic infection. By virtue of its developmental powers, which exist only in early life, the heart may be able to overcome the effect of certain lesions, e.g. myocarditis. It seems reasonable to assume that a limited lesion affecting the auricular or ventricular muscular tissue may be fully compensated for by the inherent powers of growth present at this age. Reparation in the case of developing tissues is usually more efficient than in the case of fully grown ones. On the other hand, the growth of the heart may tell adversely in the case of certain lesions such as mitral stenosis. Here the fixation of the mitral segments limits that natural growth in the size of the opening which is necessary to meet the increasing demands of the circulation. The reparative powers of the heart cannot be brought into play in the way of dilating a contracting fibrous structure, and the power of growth is limited to hypertrophy of the

left auricle and right ventricle so as to minimize the effect of the obstruction in front.

Until within recent years there seems to have been a considerable period during which there was little advance or progress made in our knowledge of heart disease and its treatment. The present writer is now dealing with his own case. The result was that interest in the subject was in abeyance. At the bedside academic discussions took place as to the nature and origin of cardiac murmurs, and where two or three were gathered together the difference of opinion was usually marked. Cases of heart disease were classified according to the valve or valves involved, and the murmur was the predominating sign. 'Backward pressure', 'compensation', and 'loss of compensation' were terms which summed up everything the user did not understand. The discovery of a cardiac irregularity led to a gloomy prognosis because of its ominous significance. Treatment was as a rule carried out in a routine manner; certain drugs had a certain reputation, some with reason and some without, but their scientific application and their suitability for various types of disease were unknown. I cannot help thinking that this state of affairs would not have been reached had the study of heart disease in early life been the student's introduction to the subject. Had this been the method there would not have been so many erroneous impressions assimilated in connexion with the signs and symptoms of cardiac disease.

A new interest in the whole subject has been aroused by the work of Dr. James Mackenzie and others. While by no means neglecting the period of youth and childhood, he has directed his attention more especially to the cardiac phenomena present in adult life. In the following pages I have tried to employ his methods in connexion with the study of cardiac disturbance and disease in earlier years. An effort has been made to study the subject *de novo*, by dropping as far as possible all preconceived ideas regarding the heart and its disorders, and by endeavouring to estimate the significance of the various signs and symptoms which are met with.

It is by no means an easy thing to give up one's preconceived ideas on any subject. Although it is true that as regards heart disease many of them may have been founded on tradition and authority only, they have nevertheless become very firmly established. It is by observing how far the views of authorities are really based on facts that one can estimate the value of these views. When tested in this way I believe it will be found that many of the current views about disturbances and diseases of the heart in childhood and youth are not founded on careful observation but on inductions and inferences with a somewhat slender basis. Dr. Mackenzie has emphasized the importance of observations carried on for a number of years on the same individuals in order to ascertain the real facts about heart disease. He himself has been fortunate in having a very large experience in this way, and

undoubtedly the mass of facts which he has collected and studied has enabled him to speak in an authoritative manner. My own opportunities for prolonged observation of individual cases have been more limited, but they have been most valuable in enabling me to draw conclusions with more precision and decision than the casual seeing of hospital patients would justify. Many of my cases have been in the families of medical men, and naturally consultations and discussions were common. When a murmur, or an irregularity, or a slight dilatation was present, heads were gravely shaken, and a gloomy prognosis was given. But why ? I believe the chief reason was that the medical mind has been led to infer that what is commonly a sign or symptom of cardiac disease in adult life is always so in childhood, and if the underlying condition is not quite clear the safest plan is to assume the worst.

Another important part of Dr. Mackenzie's work has been the analysis of the various signs of heart disturbance. For instance, many of us had become accustomed to form a more or less correct estimate of certain forms of cardiac irregularity in childhood and to say from experience that they were not of any special importance or prognostic significance. But it is a much more exact state of knowledge when an analysis of these irregularities has been made and when we can see for ourselves in tracings the youthful type of irregularity, the extra-systole, and other forms. By means of Mackenzie's ink polygraph we can make our own observations in

a manner which is more convincing and more reliable than the word of any authority. I have found the polygraph of great value in the study of heart disorders in childhood. It is an instrument which requires the expenditure of a considerable amount of time and patience to become serviceable, and there are certain limitations to its use in young children, but it will frequently prove the means of settling knotty problems at the bedside.

It is extremely probable that, in connexion with the acute forms of heart disease in early life, disorders of the rhythm of the heart may yet be discovered which are at present unsuspected. So far I do not think they have been carefully looked for by means of instruments of precision in the way that chronic heart disease has been investigated in adult life. The restlessness and dyspnœa present in acute heart disease render the investigation by means of the polygraph somewhat difficult, but in suitable cases much and valuable information may be obtained. The electro-cardiograph may also be called in for the purpose of elucidating the exact cardiac rhythm in such cases.

The analysis of signs and symptoms is but a step, although an important one, towards the interpretation of their significance. When authority lays down the law authority may be right or may be wrong. When authority speaks as the result of long experience, the opinion may be true and valuable, but the knowledge perishes with the authority in many cases because the experience is not

communicable to others. On the other hand, when the results of analysis are available generally the knowledge thus acquired is communicable to others. Irregularities of the heart, dilatation, murmurs, &c., have now been analysed and differentiated in a much more thorough manner than before. By this method of differentiation we have been placed in a much better position for estimating their significance, and for distinguishing the signs which are of no importance from those which signify real disease. It has been too much the custom, in connexion with the examination of young hearts, to assume that any sign present must have the same significance as in adult life. The whole subject of cardiac disorders and disturbances of a functional character in youth has been obscured by the assumption that there was something grave in the background which would develop in the absence of the greatest caution. It will be shown later on that those symptoms which in the case of adults are usually associated with heart disease are in the case of children much more frequently associated with disturbance of the nervous system.

Another point which has been emphasized by Dr. Mackenzie is the importance of considering the adult patient's symptoms in forming a prognosis, rather than the physical signs on cardiac examination. The great question is, what is the heart capable of doing under conditions of rest and of exercise? rather than what sort of noises it is producing. This practical test is also of extreme value

in connexion with youthful hearts which are extensively diseased or which have broken down under strain, but these form a minority of cases. In the vast majority we are dealing with acute, subacute, or chronic heart disease, and with cases of relapsing infection in which the symptoms of cardiac failure have not yet developed. We are in fact dealing in childhood and youth with the early stages of heart disease, which is of an inflammatory nature, as contrasted with heart disease in later life, which is degenerative or fibrotic in character. These early changes do not lead to dyspnœa, coughing, œdema, &c., and a prognosis founded on the absence of the latter would be unjustifiable. For this reason I think our standards in early life should be different from those established for later years. The standards, which will be considered more fully later, may be summarized here as : (1) the physical signs of endocardial, myocardial, or pericardial infection ; (2) the presence or absence of active rheumatic infection in the system ; and (3) in quiescent cases, the prospect of preventing a rheumatic reinfection. These are the factors which are of the greatest importance in estimating the prognosis of heart disease in early life.

SECTION A

CARDIAC DISTURBANCES (FUNCTIONAL)

CHAPTER II

INTRODUCTORY

The subject of cardiac disturbances in childhood is not a new one. Writing in 1865, Dr. Charles West said: 'While we should be keenly alive to the importance of every sign of heart disease, we should bear in mind that the parents not infrequently take causeless alarm at the occurrence of occasional palpitation and dyspnœa on exertion, especially if accompanied by irregularity of the pulse. Mere deranged action of the heart is by no means uncommon in children of all ages, though rarer before seven years than subsequently. It is most frequently observed in fragile, excitable children, and is not wholly amenable to direct treatment, though it subsides in the course of time under a system of general management calculated to improve the health, and of exercise judiciously regulated and always kept within such limits as not to occasion fatigue.'

Laennec wrote: 'In children more particularly the heart is perhaps always a little larger in proportion than in the adult; and many of them exhibit, in a marked degree, the stethoscopic signs

of hypertrophy or dilatation, or more commonly of both, without being at all diseased. In these persons the equilibrium is restored about the period of puberty.'

Da Costa, writing in 1890, says: 'The irregular action of the heart, its intermission, its perverted rhythm, its slow beats followed by beats hurrying to make up for the delay, are the most characteristic features of the functional cardiac disorders of childhood. Yet the degree of disturbance has not the same value attached to it as in adults; for up to about the seventh year the heart's action is often of unequal strength and rhythm, prone to be irregular in the healthiest children during sleep, and greatly influenced by the acts of breathing. But when the irregularity is marked and persistent during the waking hours and during quiet breathing, it bespeaks a cardiac disorder, except in those instances in which, joined to other foreshadowings of a cerebral malady, it points to meningeal disease.'

The heart in childhood and youth, as in adult life, is subject both to organic disease and to functional disturbance. Speaking generally, one may say that during early life organic heart disease is due to rheumatic infection, while functional disturbance is traceable to the higher nervous centres.

While careful treatment is of the utmost importance in cases of heart disease requiring it, the differentiation of such cases from those which present merely certain unusual signs or symptoms of cardiac

disturbance is equally important. As a rule, when heart disease is diagnosed in a young person the treatment adopted is such as to alter entirely the whole course of his future habits and development. If it should be that the heart is really healthy, functionally and organically, the only result is that the child's whole life is unnecessarily hampered and his development is stunted by an upbringing which is totally unsuited to a normal individual. Yet such things have happened, and are happening still, and the cause is not so much want of careful examination or ignorance of the signs of disease as a want of appreciation of the due value of the signs and symptoms which have been observed. The following case is an example.

Case I. A good many years ago a boy of six years was brought to me because after an attack of fever, thought to be malarial, a doctor in the East discovered a cardiac murmur. On examination the apex of the heart was felt pulsating a quarter of an inch outside the nipple line, and a definite short systolic apical murmur was present. I kept him under observation for six months, during which time both of the above conditions persisted. The boy had had no symptoms of any kind, and I thought we might now neglect the cardiac condition, and let him lead an ordinary life. But the parents, who had been in a state of alarm ever since they had heard of the heart murmur, considered another opinion advisable. The distinguished physician to whom he was referred decided that great precautions

must be taken, and advised that the boy be brought up under private tuition or at a special invalid school, and not allowed to indulge in any save the mildest form of exercise. The boy grew up and all his youthful energy and enthusiasm were kept in check by the numerous medical 'don'ts', which were rigidly observed. His desire for an Army career was disappointed, as his heart was not considered strong enough for the strain. When he came of age and was freed from medical control he went ahead on his own lines. He can follow the hounds and break his collar-bone with equanimity, but he will never do anything useful in the world. He has never had a symptom of cardiac disease, and his whole life has been restricted because of a blowing murmur at the apex and a slight extension of the cardiac dullness beyond what is erroneously regarded as the normal range.

I could quote other and similar cases in which the heart has been supposed to have been damaged, and in which a forecast of dangers has led to the prolonged treatment of a functionally healthy heart. Sometimes the disturbance takes the form of symptoms noticed by the friends or the patient. In other cases it is the discovery of some abnormality, by the physician, in an otherwise healthy subject which leads to a mistaken diagnosis of heart disease. In both cases there is the same erroneous line of thought, which may be expressed as follows: Certain signs and symptoms occur in connexion with and as the result of organic heart disease; some one or other

of these is present in this young person ; therefore this young person is the subject of heart disease. When thus expressed the error is manifest, but there are probably few of us who have not employed this line of reasoning at some time or another.

It is to be noted that there is a great similarity between the cardiac signs and the cardiac symptoms of both functional disturbance and organic disease. We meet with irregularities, murmurs, dilatation, faintness, shortness of breath, &c., in both. I shall endeavour to make the differentiation as clear as possible. In some cases this can be demonstrated positively, and in others one can only indicate certain aids in the diagnosis.

CHAPTER III

CARDIAC IRREGULARITY OR ARRYTHMIA

(A) Sinus Irregularities, (B) Extra-systoles, (C) Compound Irregularities

There are various forms of cardiac irregularity in the young, and the full significance of these will not be grasped unless one is familiar with the normal variations of cardiac action in early life.

(*a*) Sinus Irregularities

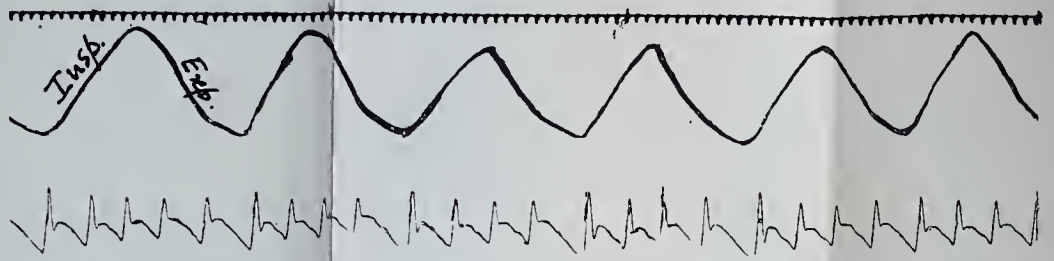
The first great class is embraced under the term 'sinus irregularities'. They are so called because

they originate at the sinus venosus, in the sino-auricular node, which is the normal 'pacemaker' of the heart. They are also termed 'vagal irregularities' because they are dependent on irregular action of the vagus, which is the chief regulator of the cardiac rate. In a radial tracing one sees that the striking feature is the difference in the periods of diastole, so that the result of a sinus irregularity may be described as a diastolic variation. As the impulse starts at the normal place, the sino-auricular node, the contractions of the various chambers of the heart take place in their regular sequence, i.e. the intra-cardiac rhythm is not disturbed. So characteristic of early life is this form of irregularity that Mackenzie has termed it 'the youthful type of irregularity'. It is very frequently met with throughout the first two decades of life. Mackenzie has further emphasized the influence which respiration plays in bringing about this irregular action of the vagus. In my experience, so marked is this cardio-respiratory association that one cannot doubt that respiration is the chief factor in the production of the vast majority of cases of sinus irregularities. With this form I propose to deal first of all.

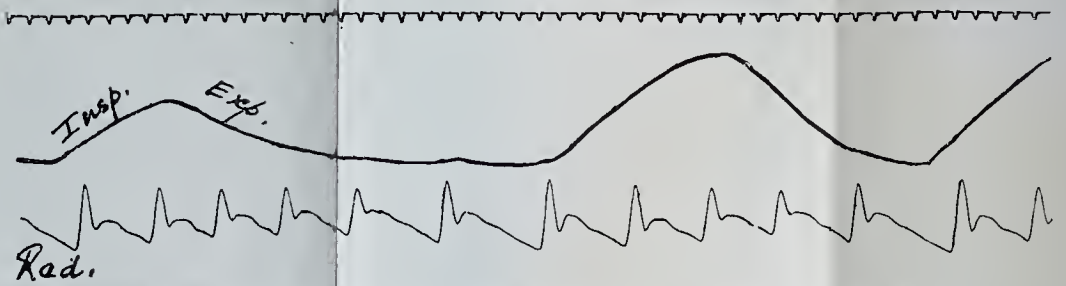
Clinically considered, the features of this form of irregularity are as follows: One can detect on feeling the pulse in a great number of young people that the rate varies, a few quicker beats being followed by a few slower ones. This is so common that it has come to be recognized as natural and physiological, and it has the same basis as the more

striking forms which have sometimes been regarded as serious irregularities. Again, we may detect a long pause interrupting at intervals the ordinary beats, and this may be put down as an 'intermittent' pulse. Or again, we may find the pulse so irregular as regards the force and timing of the beats that it can only be described as a marked irregularity. This last condition may be due solely to a sinus irregularity associated with respiration, or it may be due to a combination of forms, as will be shown later.

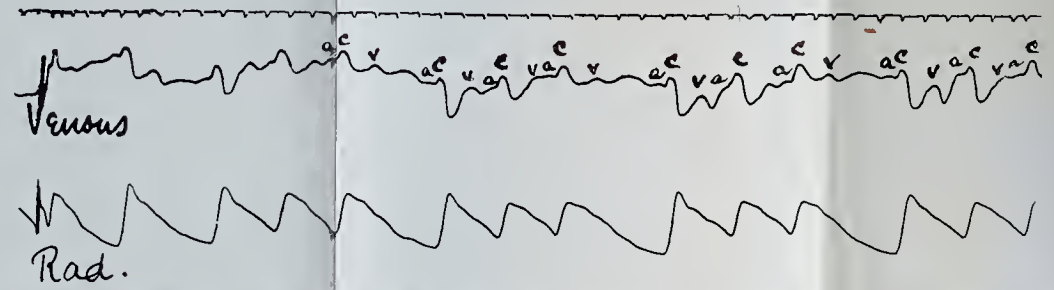
On auscultation of the heart, the sounds are clear and the second always follows the first at the normal interval, but the diastolic periods vary. Sometimes a few quick beats occur of a weak character followed by a few slower of stronger character, or a long pause representing a standstill of the whole heart comes at intervals, or the intervals seem to be markedly irregular. While auscultating it may be possible to trace definitely the association of the cardiac irregularity with the respiratory movements. During inspiration there is a quickening of the cardiac rate, and during expiration there is a slowing. If it is ordinary quiet breathing that is going on one may be able to time the quicker and slower periods with the acts of inspiration and expiration. If the patient is breathing slowly and deeply we shall find this timing much more easy and the irregularity probably well marked. If he is breathing quickly we shall find the timing more difficult and the irregularity less marked; while if he holds his



IG. 1. Cardio-respiratory irregularity (youthful type). Healthy boy of 11 years. Breathing regular. Radial tracing shows marked slowing of pulse rate during expiration, due to prolongation of diastolic period. Pulse rate = 75.



IG. 2. Cardio-respiratory irregularity (youthful type). Same case as above. Breathing irregular. Marked slowing of pulse rate during expiration, due to prolongation of diastole. Pulse rate = 75.



G. 3. 'Intermittent pulse.' Healthy girl of 6 years. The apparent intermittency occurs with great regularity after y third beat of the pulse. The venous tracing (upper) shows that the whole heart took part in the long pauses or intermissions. e rate = 82.

To face p. 19.

breath for a time, we shall find that the irregularity of the heart disappears entirely during the period of apnœa.

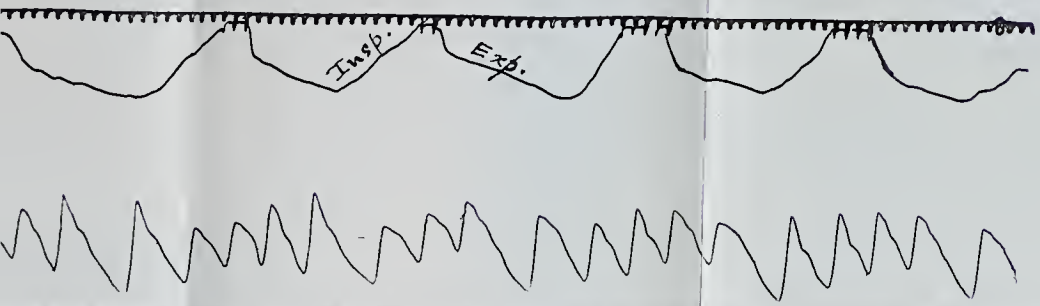
The exact nature of this cardio-respiratory irregularity is best demonstrated by means of tracings. In Fig. 1, which is taken from a perfectly healthy boy of eleven years, the respiratory tracing shows regular breathing, while the radial tracing shows an equally regular lengthening of diastole at the end of expiration. Viewed in this light the radial irregularity is perfectly regular in its period of occurrence. When the breathing becomes more irregular, as in Fig. 2, the variations in the length of diastole also become more irregular, but have clearly the same association. It is to be noted that the pulse is stronger after the longer pauses than after the briefer ones, but this is merely the result of the physiological law that the left ventricle when beating slowly will contract more strongly than when acting quickly. In the above case this difference in the force of the pulse-beats could not be detected clinically, and on auscultation of the heart all that could be determined was a slight variation in rate from time to time.

The 'intermittent' form of pulse, or what will be recognized clinically as such, is shown in Fig. 3. These tracings were taken from a girl of six years who was sent to me because of an irregular action of the heart. On feeling the pulse one could recognize a definite intermission at fairly regular intervals, the nature of which was not at first clear. The

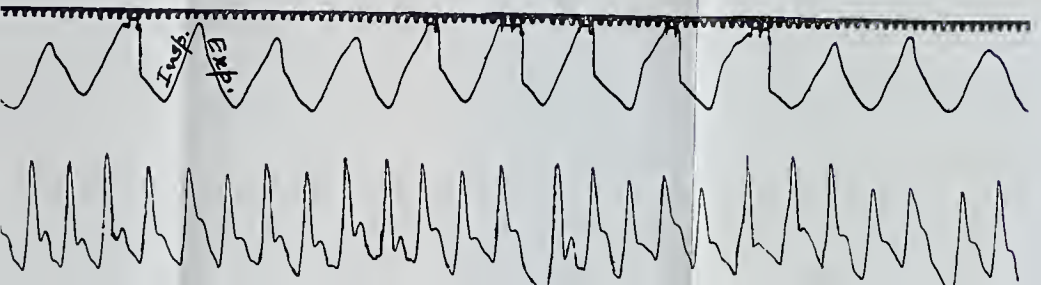
radial tracing showed this intermittent action very well. A venous tracing showed that the whole heart took part in the irregularity. It was clearly a case of sinus irregularity. A combined cardio-respiratory tracing (Fig. 4) demonstrated that the intermittency was really due to slowing of the cardiac rate at the end of expiration, there being usually at this period one much longer pause than at any other time.

Sometimes the degree of irregularity is so marked as to be very puzzling. In the following case I was unable to tell the form of irregularity from feeling the pulse, and was led to suspect it from auscultation of the heart. A boy of twelve years was admitted to hospital because of fits, delirium, and irregular action of the heart. His fits were cured and his moral behaviour was much improved by firm compression of the supra-orbital nerve, but the cardiac irregularity persisted. The tracing of the radial (Fig. 5) shows a marked degree of irregularity. When this is compared with the respiratory tracing above, the explanation becomes clearer, for we see the association of the various respiratory movements with the various changes in the pulse. There is a well-marked increase of the cardiac rate during inspiration and a slowing during expiration. The boy was asked to breathe quickly, and the next tracing (Fig. 6) shows the result. There is little of the irregularity left, and none was to be felt when testing the pulse with the finger. He was next asked to hold his breath, and the tracing again

ittent pulse.' Same case as Fig. 3. This cardio-respiratory tracing shows that the long pauses or intermissions occur with uring expiration only, and that the variations in the cardiac rate are clearly respiratory in origin. Pulse rate = 90.



-respiratory irregularity. Boy of 12 years. The radial pulse shows a marked irregularity in the force and duration of hing slow and regular. The respiratory tracing shows that the quicker and smaller radial beat occurs during inspiration, d stronger beat during expiration. (Heart healthy.) Pulse rate = 70.



showed the disappearance of the irregularity. When breathing was resumed one saw at once, as shown in Fig. 7, the onset of the irregularity in the pulse, which was clearly respiratory in origin. These tests were conclusive as to the nature of the pulse irregularity.

It is often stated that cardiac irregularity is common in early life, quite apart from any disease,

FIG. 7. Effect of holding the breath on cardio-respiratory irregularity. Same case. Boy was told to hold his breath and the marked radial irregularity disappeared, to recur at the first inspiration.

during sleep, and this fact may be easily confirmed on examination. It is not so often stated what the form of irregularity is, and what it is due to. Another fact that may be easily confirmed is the irregular character of the respiration in early life during sleep. My own investigations have shown that when the pulse was irregular during sleep, the breathing was also irregular, and further, that the irregularity of the pulse was of the character we are now considering, namely, directly associated with respiration. If the breathing during sleep is slow

and deep the respiratory effect on the pulse will probably be manifest.

The variations in the occurrence of this irregularity from time to time may be very puzzling, and the fact is to be associated with the variations that take place in the breathing during early life. The determination of the youthful type of irregularity may depend entirely on close observation of the

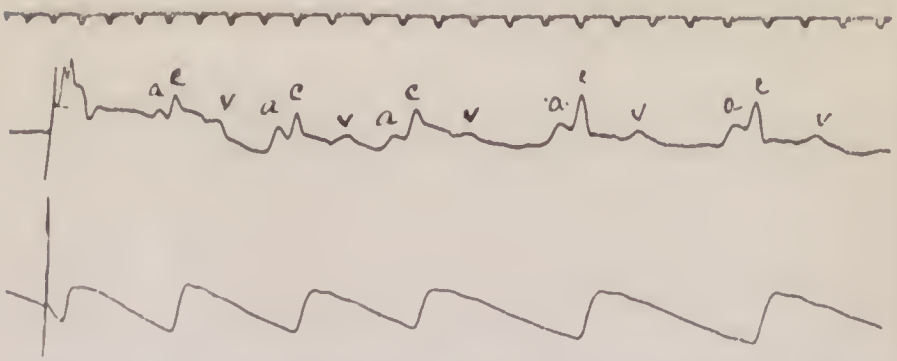
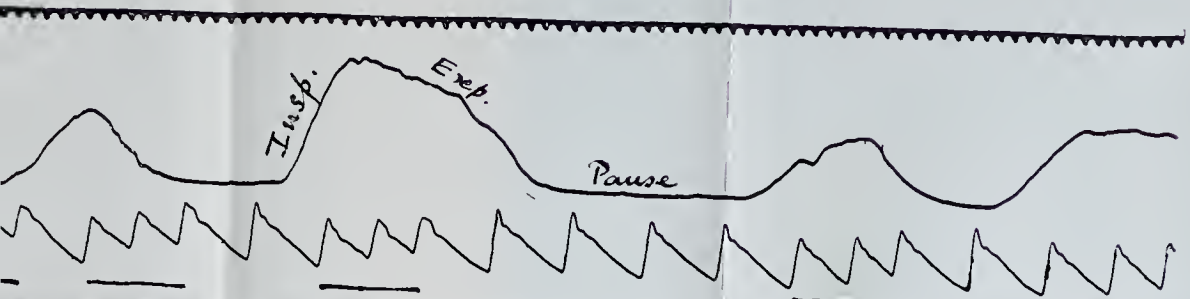
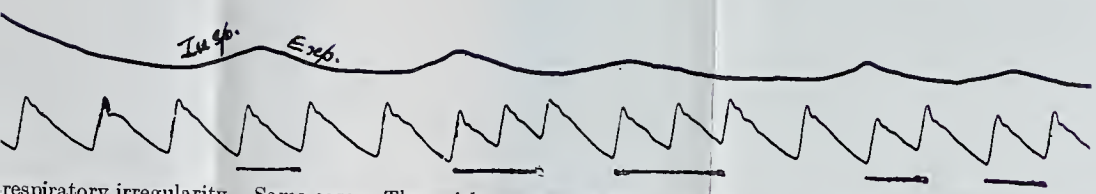


FIG. 8. Cardio-respiratory irregularity. Healthy boy of 8 years. Irregularity of pulse felt, and shown by radial tracing to consist of varying length in diastole. The jugular pulse (upper tracing) was normal, save for the same diastolic variation, showing that the whole heart shared in the irregularity. Pulse rate = 70.

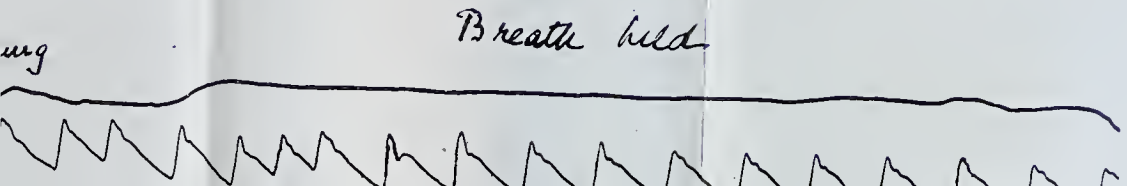
breathing, and in some cases tracings may be necessary. In the case of a boy of eight years the pulse at times showed a very slight degree of respiratory variation (Fig. 8) and a normal tracing was obtained. At other times the pulse was so irregular (Fig. 9) that clinically one could not decide the nature of the irregularity. The accompanying respiratory tracing shows very irregular breathing, and also that each respiration has a distinct effect on the



respiratory irregularity. Same case as Fig. 8. At times the pulse was so irregular as to be puzzling (radial tracing). y tracing was taken simultaneously, it was seen that the breathing was also irregular, and that the respirations were ac rate. A well-marked example of the youthful type of irregularity. Pulse rate = 70.



respiratory irregularity. Same case. The quick response of the heart to each respiratory movement, however slight, is shown in the radial tracing.



pulse. This figure also shows how the depth and duration of each respiration may have a corresponding influence on the cardiac action in the matter of irregularity-production. Fig. 10 further shows how the slightest respiratory act may have an immediate effect in altering the cardiac rate; while the last tracing (Fig. 11) shows that with the cessation of respiration (breath held) the cardiac action becomes quite regular. In many cases such as the above it will be possible to tell from the pulse tracing what the character of the breathing has been at the same time, as to rate, depth, and regularity.

We have next to consider **the conditions which are likely to induce this type of irregularity,** or in which it is likely to occur. We are still dealing with cases which present no sign of cardiac disease. The youthful type of irregularity is associated in what may be termed its clinical or recognizable form with a slow pulse. *Per contra*, when the pulse rate is increased from any cause, excitement, fever, exertion, &c., it disappears entirely or becomes much less marked. This irregularity is often present with the slow pulse which follows an acute illness, e.g. influenza, measles, &c. There is a class of young patients whom I shall refer to as neurasthenics, and whom Mackenzie would describe as suffering from the X-disease, in whom this youthful irregularity is common. The underlying cause is clearly the instability of the central nervous system, and in this instability the vagal centre shares. So marked have I found this association that when in an otherwise

healthy young person the youthful type of irregularity is clinically pronounced I have come to look on it as a sign of general nervous instability. This will be referred to again later. There will still remain

FIG. 12. Cardio-respiratory irregularity in cerebral disease. Boy of 6 years, suffering from cerebral abscess, and semi-comatose. Respiration = 20. Pulse = 78. The radial tracing shows a marked irregularity due to variations in the length of the diastolic periods.

FIG. 13. Cardio-respiratory irregularity. Same case. When a respiratory tracing was taken along with the radial, it was shown that the irregularity of the pulse was respiratory in origin, i.e. the youthful type of irregularity. The quickening was inspiratory and the slowing was expiratory.

a large number of young persons in whom this irregularity is present without any obvious reason.

In cerebral disease and disorder an irregular type of pulse has often been noted, and so far as my observations have gone the respiratory factor is the most common cause. A slow and irregular pulse is characteristic of many cases of meningitis, and if the

FIG. 14. Cardio-respiratory irregularity in cerebral disease. Child of two years suffering from tuberculous meningitis. Cheyne-Stokes respiration. Tracings taken twenty-four hours before death. A cycle of respirations leads to an acceleration of the cardiac rate, while during the period of apnœa the heart slows down.

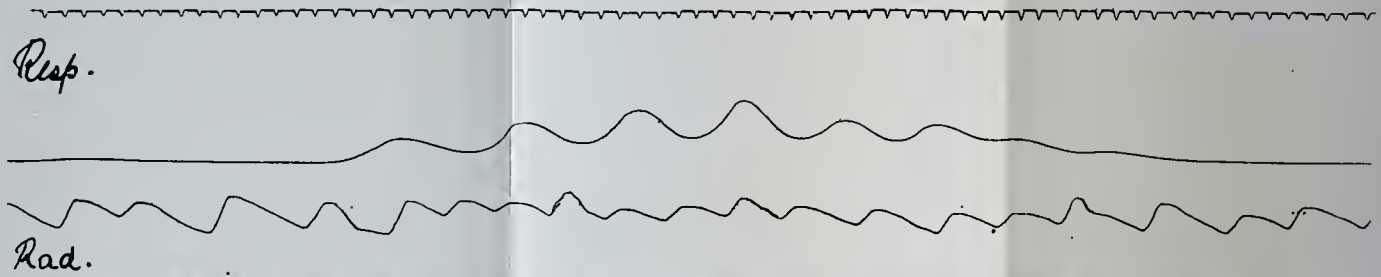


FIG. 15. Cardio-respiratory irregularity in cerebral disease. Same case as above. Tracing more detailed. Shows same diastolic variation as above.

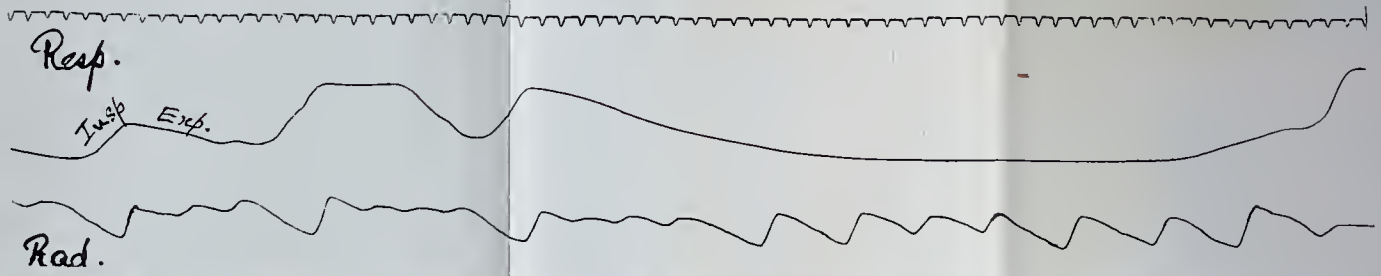


FIG. 16. Cardio-respiratory irregularity in cerebral disease. Same case as above. Breathing irregularly cyclic. Youthful type of irregularity is seen in the radial tracing, where the rate of the pulse is shown to be accelerated during inspiration, slowed during expiration, and rendered much more regular during period of apnœa. Pulse rate during respiratory movement = 100 per minute; pulse rate during apnœic period = 70 per minute.

cardiac action is rapid the irregularity is not usually present. As the breathing is also often irregular it may be very difficult, in the absence of graphic records of the pulse and respiration, to determine the exact form of cardiac irregularity which is present. We may find, as in Fig. 12, that there is merely a diastolic variation which suggests a respiratory source, and which is shown to have this origin when a respiratory tracing is combined (Fig. 13). These tracings were taken from a boy of six years who was in a semi-comatose condition as the result of pressure from a cerebral abscess. When the breathing is of a cyclic or Cheyne-Stokes type the effects on the regularity of the pulse are more complicated. Tracings were obtained from a child of two years shortly before death from tuberculous meningitis. A cycle of respirations led to an acceleration of the cardiac rate, followed by a slowing down during the period of apnœa, as shown in Fig. 14. This is the usual accompaniment of this type of breathing. When the details are brought out further (Fig. 15) it will be seen that the diastolic variation is present even during a cycle of respiration. When the breathing becomes markedly irregular (Fig. 16) it will be seen how the characteristics of the youthful type of cardiac irregularity are shown in the radial tracing. I mention these facts in connexion with cerebral disease because they have a bearing on the ætiology of the youthful type of cardiac irregularity in healthy young people.

Passing now to the explanation of this type of

irregularity we find that all the evidence points to its being due to instability of the vagal centre. It is to be noted that in childhood the vagal centres for the heart and for respiration are not yet fully under the control of the higher inhibitory centres, and have not firmly established a regular automatic action. Some years ago I had occasion to consult the late Dr. Hughlings Jackson about certain nervous phenomena in children, and he told me he did not like to draw conclusions from the action of the nervous system in childhood because it was not yet 'a going concern'. The vagal centre in infancy and childhood is not a going concern, but is subject to various disturbing influences, both from within and from without, which easily upset the regularity of its action. Hence comes the youthful type of cardiac irregularity. The age at which control of the vagal centres is developed varies in different individuals, so that we may find this form of irregularity present at any age up to adult life. When any general instability of the nervous system is present it may show itself markedly in this altered vagal action. When any cerebral disease is present it may act in a similar manner, and I think it will be found that the irregular action of the heart in diseases like tuberculous meningitis is due primarily to vagal instability and secondarily to the influence of respiration on the vagal centre.

Lewis has determined the presence of this vagal irregularity in newly-born infants by means of the electro-cardiograph. Mackenzie says that probably

every healthy individual may show it at one time or another, and that it is not uncommon between the ages of 20 and 30 years, after which it becomes rarer. It is commonly present in dogs, and in them the heart has been carefully examined post-mortem, and found to be structurally healthy. It can usually be elicited in the case of normal and healthy children by making them breathe deeply and slowly. It can be abolished by a hypodermic injection of atropine, which paralyses the vagus, and in dogs by section of the vagi.

The diagnosis of this type of irregularity can usually be made with accuracy if the observer will bear in mind the association with respiration. The character of the irregularity may be suspected on feeling the pulse, and told more clearly by auscultation of the heart. The distinguishing features, its disappearance on rapid breathing or on holding the breath, are usually easily elicited. As a final test, not usually necessary, graphic records of the pulse and respiration may be taken. The fact that venous pulse, arterial pulse, and ventricular contraction all share in the irregularity, which corresponds with respiratory movements, is quite diagnostic. From what has been stated it will be seen that no significance is to be attached to this type of irregularity *per se*. It is not a symptom of heart disease, nor is it in any way associated with heart disease. In itself it has no influence on the heart, or the health, or the future prospects of the patient. The presence of the irregularity is not usually manifested to the

patient by any symptoms. The only danger associated with it lies in its discovery by people who do not appreciate its significance, and who may base a gloomy prognosis of heart disease on its presence. As the prognosis is always favourable apart from any treatment this irregularity is to be regarded as a symptom which does not call for any treatment.

As regards other forms of sinus irregularity, apart from the respiratory, it has to be remembered that the vagus may be influenced by other forms of local or reflex disturbance, producing a cardiac irregularity marked by diastolic variations. Lewis has found an irregularity in the whole heart of mild degree, in which shorter and longer pauses are mingled indiscriminately, not infrequently in quite young and apparently healthy children, and without any relation to respiration. I have not found any such cases presenting a pulse irregularity sufficient to attract attention clinically. The rule applies in all cases of sinus irregularity that so far as the heart is concerned the condition may be ignored.

(b) Extra-systoles

An extra-systole is the term applied to a premature contraction of the heart. It may be convenient to adopt Mackenzie's suggestion that 'the term "extra-systole" should be limited to those premature contractions of auricle or ventricle in response to a stimulus from some abnormal point of the heart, but where, otherwise, the fundamental or sinus rhythm of the heart is maintained'. Extra-systoles

are classified according to their site of origin as auricular, ventricular, &c. Their mode of origin and pathology have led to much discussion, and their elucidation has been greatly helped by the more recent methods of investigation, but for information on these subjects the reader must consult the newer text-books. While the venous tracings of extra-systoles are of extreme value they also are compli-

Fig. 17. Extra-systoles or premature contractions. In radial tracing two are shown. They occur prematurely, are weaker than an ordinary beat, and are followed by a longer than usual pause and a stronger than usual beat. They bear no relation to the respiratory movements, and occur independently of any diastolic variation due to that cause.

cated, and I shall only refer to them so far as is necessary for my clinical purpose.

It has come to be recognized that extra-systoles may occur at all ages, most frequently in association with evidences of organic heart disease, but also as an isolated phenomenon. It is with their occurrence in those who present no other signs of heart disease that we are now dealing. The recognition of extra-systoles is important because they lead to an irregularity of the pulse and of the heart which may excite surprise and, as has often been shown in the

past, even alarm. In the first decade of life extra-systoles, although common enough as an occasional occurrence, are not often present as a definite form of pulse irregularity ; in the second decade they are more frequently met with.

Although a clear demonstration of their nature is best supplied by graphic records, their clinical recognition is usually easy by the ordinary bedside methods of examination. As a rule, in my experience, young people do not mention any subjective symptoms from the presence of extra-systoles. The change noted in the pulse depends on the strength of the ventricular extra-systole, which from its predominance in frequency over all other forms is the only one we need consider here. If the premature contraction of the ventricle is sufficiently strong to open the aortic valve, the result will be transmitted and felt at the wrist ; if it is not, the stream of blood in the arteries will not be affected. Palpation of the heart is very useful in detecting extra-systoles, especially in early life when the chest wall is thin. One can feel at the apex soon after a normal beat a second one, at an interval shorter than normal from the preceding one, and followed by a pause longer than normal. Sometimes this extra-systole feels stronger than a normal beat. On auscultation one recognizes an extra-systole by the occurrence after the two normal sounds of a third sound, at a short interval (prematurely), and if the aortic valve has been opened of a fourth sound. This is a form of double beat which is usually followed by

a pause perceptibly longer than the normal diastole. These phenomena may be reproduced in connexion with every few beats of the heart, at regular or irregular intervals, or only as an occasional phenomenon. When one examines the pulse there may be a double beat, the second following the first at a short interval, and being followed by a pause longer than usual. If the blood has not been driven through the aortic valve, the extra-systole is represented at the radial pulse by a long pause. According to which of these conditions is present the pulse

FIG. 18. Extra-systoles. Healthy boy of 9 years. A marked clinical irregularity of the pulse present, shown by the tracing to be due to a succession of extra-systoles, occurring at every third or fourth beat. The premature character and weakness of the beat are shown, and also the long pause which follows it.

irregularity in connexion with extra-systoles may be bigeminal in character, or intermittent (missed beat) as interpreted clinically.

Tracings explain these signs clearly. In Fig. 18 a succession of extra-systoles is shown, occurring at every third or fourth beat, and each extra-systole has reached the wrist. The premature character and weakness of the beat, and the long pause which follows it, are shown. This pulse would be regarded as one of a bigeminal character, but irregularly so. In the next tracing (Fig. 19) a similar condition is shown in a more expanded form. In Fig. 20 there is seen a tracing of a pulse which is at times

bigeminal and at times intermittent. The tracing begins with a succession of extra-systoles, some of which reach the wrist (•) and some do not (×), a somewhat confused irregularity which could not be recognized very easily on feeling the pulse. On comparing the radial tracing with the cardiogram above, it is seen that each extra-systole is recorded there whether it reaches the wrist or not. I give some other tracings from young patients which show

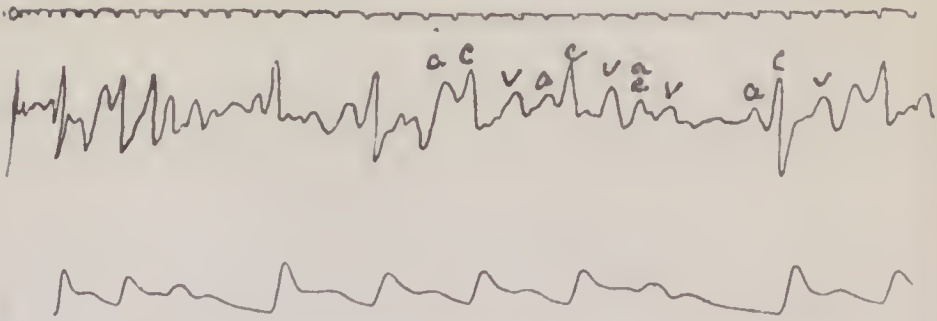


Fig. 19. Extra-systoles. Same as last in more detailed tracing. Upper part is the venous tracing.

the extreme irregularity met with in connexion with extra-systoles (Fig. 21) and one which shows the venous pulse in such a condition (Fig. 19). On comparing these tracings with those taken from cases of organic heart disease presenting extra-systoles, I cannot find any points of difference by which they can be distinguished.

Case II. Cardiac trouble was suspected in a boy of ten years because he had fallen down three times in the course of a few weeks, while playing, on the floor. When he was four years old the doctor had

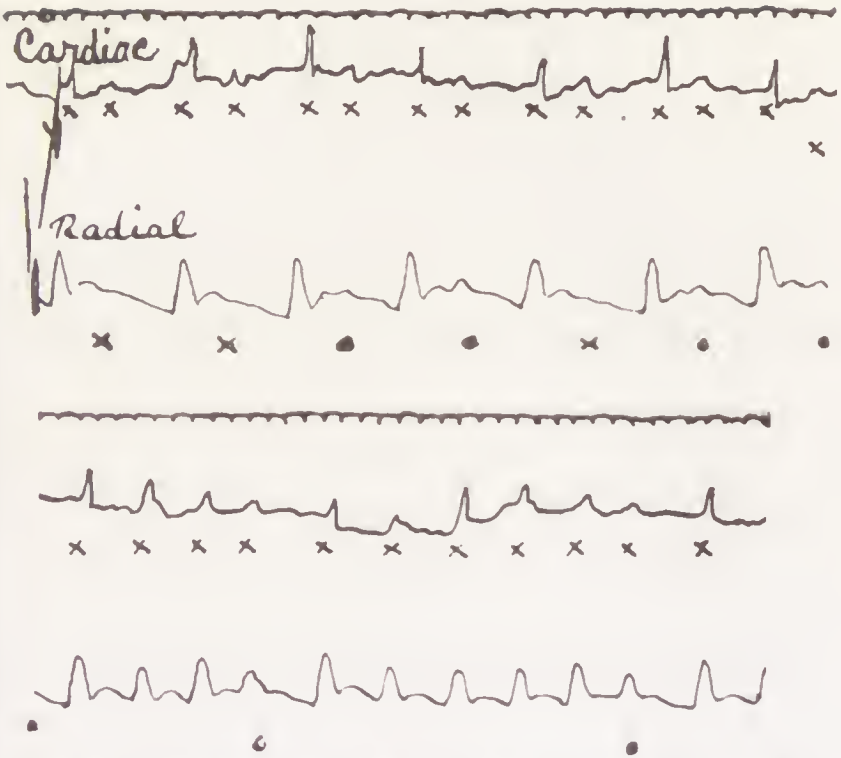


FIG. 20. Extra-systoles. Healthy boy of 10 years. Clinically the pulse felt at times bigeminal, and at times intermittent. The radial tracing shows a series of extra-systoles, some of which reach the wrist (marked •) and some do not (marked ×). That these are extra-systoles and not real intermissions is shown by the cardiac tracing above, where each heart-beat (marked ×) is recorded whether it affects the pulse at the wrist or not. In the second half of the figure, which is continuous with the first, the cardiac action is seen to be more regular, and the extra-systoles less frequent.

discovered a marked irregularity of the pulse and heart, and this had usually been found to be present ever since when examined for. He had had no other symptoms of heart disease, and had never noticed

the irregularity referred to by any subjective symptoms. His previous health had been good, the boy never having had any serious illness or any manifestations of rheumatic infection.

He was a tall, thin, and rather anæmic youth, and was described by his mother as distinctly of a nervous and excitable temperament. His muscular powers, cardiac and otherwise, seemed to be good from the fact that he had taken the prizes at school for running and jumping during the last term.

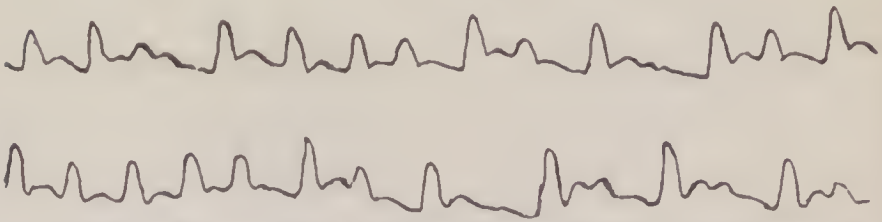


Fig. 21. Extra-systoles. Showing irregular forms of pulse tracing which may be produced. The exact condition is not always easily recognized clinically.

The cardiac action was very irregular. The apex beat was situated a quarter of an inch outside the nipple line in the 4th space. At the apex both sounds were audible, and a soft murmur, systolic in time, was heard on light pressure with the stethoscope, more marked on firm pressure. The murmur was not conducted outwards, but was audible over the præcordium as far as the pulmonary area. The pulse rate was 95, and while at times there was a succession of regular beats, the predominating feature was an irregularity of a very marked character. The urine gave a marked reaction for nucleo-

albumin, but not for serum-albumin. Pulse tracings showed the presence of extra-systoles and no other form of irregularity.

The boy was ordered general tonic and hygienic treatment. When seen a week later he was looking better and brighter—the result of a week's freedom and open-air life. The apex beat was in the nipple line, the only murmur heard was a short systolic whiff in the pulmonary area. The cardiac action was perfectly regular, and a long tracing of the radial artery failed to reveal the presence of a single extra-systole.

Although the second examination was useful in confirming the conclusions drawn from the first, it was clear from the latter that the boy had no signs of heart disease, and that the fainting and irregularity were to be ascribed to other causes. The fact that the extra-systoles in the form of an irregular pulse had been noted since the age of four years was in itself suggestive of a functional disturbance. The various forms of local instability present in this patient were regarded as dependent on the general instability of his developing nervous system. A year later it was reported that the boy was quite well, but that the cardiac irregularity was still present. No treatment directed to the heart had been adopted.

One of the striking features about the extra-systole is its fleeting character. It may be discovered casually on one examination of a patient and then cease entirely. Its presence may have led to a diagnosis of cardiac irregularity, and when

a consultation takes place the heart is found to be perfectly regular in its action. This points to the fact, which is a matter of everyday observation, that in susceptible young people extra-systoles are easily excited and easily made to disappear. They are most conspicuously present when the heart is acting slowly, and if the rate is increased to above 100, as by fever, exercise, or emotion, they will probably disappear. Change of posture from the upright to the recumbent position may lead to their cessation. The exciting factor is often difficult to trace, but I believe that in young people extra-systoles often occur as the result of gastro-intestinal disturbance, e.g. dilated stomach, undigested food, &c., which acts reflexly on the heart. Like other forms of functional irregularity they are most commonly present in neurotic children whose nervous systems are prolific of reflex disturbances. As a rule also it is only those introspective young people who observe closely their own sensations who complain of the presence of extra-systoles. They may refer to a stopping of the heart or a fluttering of the heart, which is clearly a manifestation of the extra-systole. In the convalescent stage of acute illness, e.g. pneumonia, I have frequently found extra-systoles occur, usually at long intervals, and without any sign of cardiac disease. Finally cases arise in which extra-systoles are found without any exciting cause which can be discovered.

The significance of extra-systoles occurring in connexion with apparently sound hearts has not yet

been definitely settled by general consent. Lewis says that 'While premature contractions have unquestionably a relatively insignificant import, as compared to many forms of cardiac irregularity, entire neglect of their presence is not advisable'. Wenckebach states that 'in themselves extra-systoles possess no diagnostic significance, and no conclusions can be drawn from their mere presence as to any form of cardiac disease; all that they prove is that the heart is disturbed in its rhythm, and stimulated to extra-contractions by abnormal agencies'. He thinks there is some abnormally high excitability of the heart, either congenital or acquired, which is specially manifested in the great class of neurasthenics. Mackenzie says 'it will be found that extra-systoles in themselves are not signs of any specific injury to the heart, nor should a prognosis of any gravity be based on their appearance alone. I have watched individuals for over twenty-five years who have presented extra-systoles, sometimes with greater frequency than at other times; and these people have led laborious lives, and have never shown the slightest symptoms of heart failure, or any other evidence of heart impairment. I have watched young people grow into manhood and lead vigorous lives'.

As the exact ætiology of extra-systoles is unknown prognosis should be based on the result of experience. In the past it has too often been the custom to regard extra-systoles, or the irregularity of the heart caused by extra-systoles, as a sign of cardiac impair-

ment, a rather serious prognosis has been given, and restrictive treatment or cardiac treatment has been ordered. But experience has shown that these lines of thought and procedure are neither necessary nor advisable. In the light of present-day knowledge of the mechanism involved in the production of extra-systoles it is incumbent on those who give a serious prognosis or a guarded prognosis to bring forward evidence of any injurious result quâ heart or patient from their presence. So far as I know no such evidence has been forthcoming. Mackenzie speaks from a prolonged observation of cases of extra-systole. My own more limited experience entirely confirms his conclusions. As regards the treatment of the class of case under consideration, that showing extra-systoles and no evidence of organic heart disease or active rheumatic infection, I have simply ignored the cardiac irregularity and let the young subjects indulge in every form of exercise without restriction. I have not seen any bad results, nor have I found any evidence that the extra-systoles had any bearing whatever on the patient's physical condition.

In a number of my cases the cardiac irregularity, which was afterwards shown to be due to extra-systoles, had been present from a very early age, viz. the third or fourth year. Poynton has met with extra-systoles in a female child of two years, who had suffered from 'fits', but had no sign of heart disease. Lewis was able to demonstrate in this case by means of the electro-cardiograph that

the irregularity was due to auricular extra-systoles. If a prognosis is to be based on ascertained facts, as it ought to be, in the case of extra-systoles occurring in healthy children all the facts are favourable. In deciding as to treatment I think one may confidently ignore the existence of the irregularity from the cardiac standpoint, and direct attention to any other part of the body which may require toning up. In my experience this in the case of young people will usually be found in an unstable nervous system.

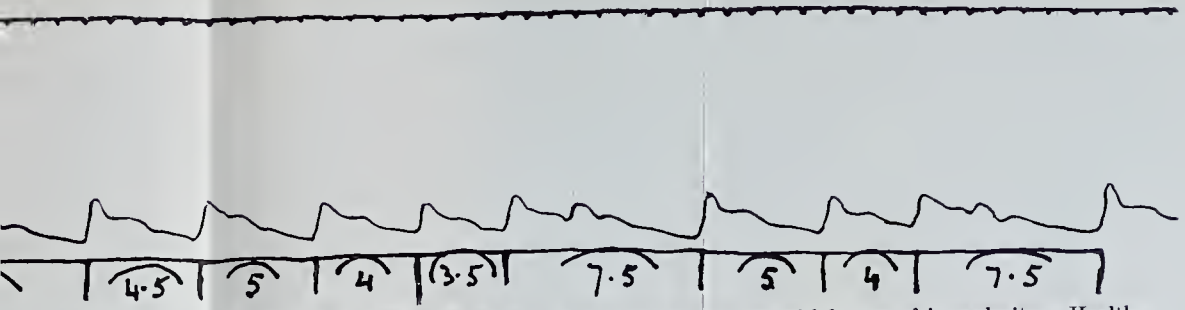
(c) Compound Irregularities

While in cases of organic heart disease a mixed form of irregularity of the cardiac action is not uncommon, in the functional disturbances one definite type is usually present. Yet so common amongst all children is the respiratory variation of heart rate that this may tend to confuse an observer when it is present along with another form of irregularity. While ordinary clinical methods will serve to distinguish between respiratory irregularity and extra-systoles when occurring separately, it may be necessary to take graphic records to elucidate the conditions when they occur at the same time in the same patient. As a diagnostic point it may be suggested that if the clinical observer, bearing in mind the frequency of respiratory variation, will make the patient hold his breath, that form of irregularity will disappear and the second one can be studied separately.

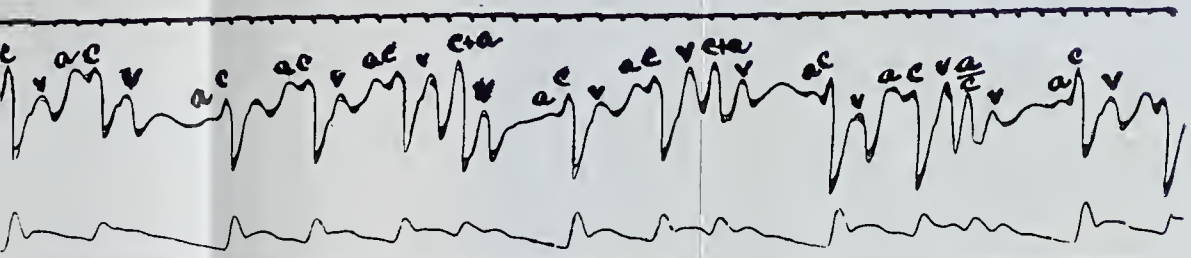
Case III. A boy of nine years was seen because of an irregular action of the heart. This had been noted three years previously and had persisted. There had been no evidences of cardiac infection. He was a healthy-looking boy, and the only complaint was of indigestion and occasional attacks of urticaria and herpes. The action of the heart was markedly irregular. Extra-systoles could be detected, and also intermittent action. Otherwise no signs or symptoms of cardiac disease could be detected.

A radial tracing (Fig. 22) showed the presence of ventricular extra-systoles occurring at irregular intervals. In addition it was apparent that apart from the extra-systoles the diastolic periods accompanying normal beats varied greatly in length. A venous tracing (Fig. 23) showed the influence of the extra-systoles, but was otherwise normal. A combined respiratory and radial tracing (Fig. 24) showed that the long diastolic intervals came towards the end of respiration, while the shorter ones followed immediately on inspiration. The extra-systoles occurred at any time during the respiratory cycle and were clearly not affected by it.

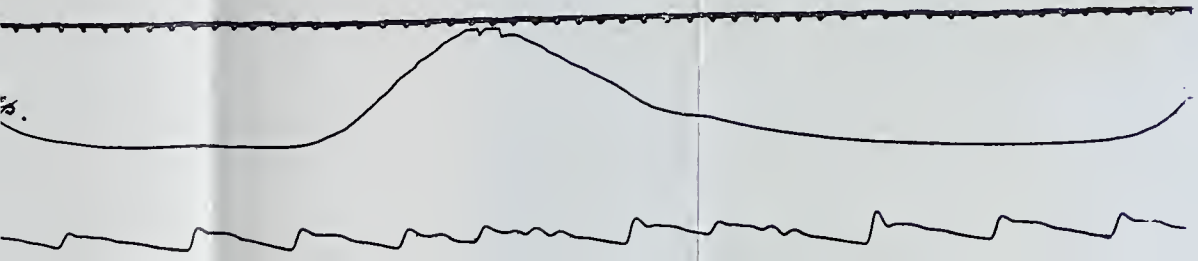
The clinical problem of the cardiac irregularity was thus shown to be dependent on two different factors. As each of these was of no significance by itself, it was concluded that the combination of the two was of no importance. Advice was given on these lines and the boy has continued to live an ordinary life, without any alteration so far in the



nd irregularity, i.e. a mixture of two or more forms. Extra-systoles and youthful type of irregularity. Healthy dial tracing shows extra-systoles at intervals. In addition there is a variation in the diastolic periods of the ordinary in fifths of a second, 3·5, some 4, some 4·5, and some 5. The pulse rate is 70 per minute.



nd irregularity. Same case as above. The upper (venous) tracing shows that at the extra-systoles auricular con- beat occur at the same time ($c+a$). It also shows that the whole heart shares in the varying length of the diastolic auses correspond with pauses in venous tracing.



irregularity but without any development of further cardiac signs.

It may sometimes be noted that this respiratory form of cardiac irregularity in a marked clinical form is a family tendency.

Case IV. The brother of the last case was seen at the age of three years because of some weakness in the lower extremities, and the doctor wanted also my opinion as to an irregular action of the heart which he had noticed. The boy had had no cardiac symptoms, and no cardiac signs apart from the irregularity had been observed. The irregularity took the form of a few hurried beats followed by one or two pauses like intermissions in the cardiac action. It was also quite marked on feeling the pulse. At the time I did not know the exact form of irregularity present, but was familiar with the type as common in childhood and believed it of no prognostic significance.

The boy was seen again four years later. The irregularity was still present and of the same character. The heart was otherwise normal in every way and there were no cardiac symptoms. A tracing (Fig. 25) showed that the irregularity was entirely respiratory in character, there being a marked increase in the rate on inspiration, and a marked slowing during respiration. When the boy held his breath the irregularity disappeared.

Another form of compound irregularity may be briefly mentioned. Reference has been made to an irregularity in the force and timing of the beats in

connexion with the respiratory variation. As a rule it is the irregular timing which attracts notice. In some cases, however, one may be struck by the variation in the force of the beats. It may be found that a few small beats are followed by a few stronger ones, and the explanation may at first appear to be obscure. The condition suggests that designated as the *pulsus paradoxus*, in which the pulse dies away

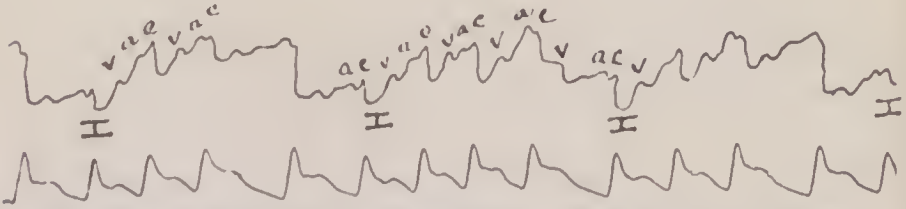
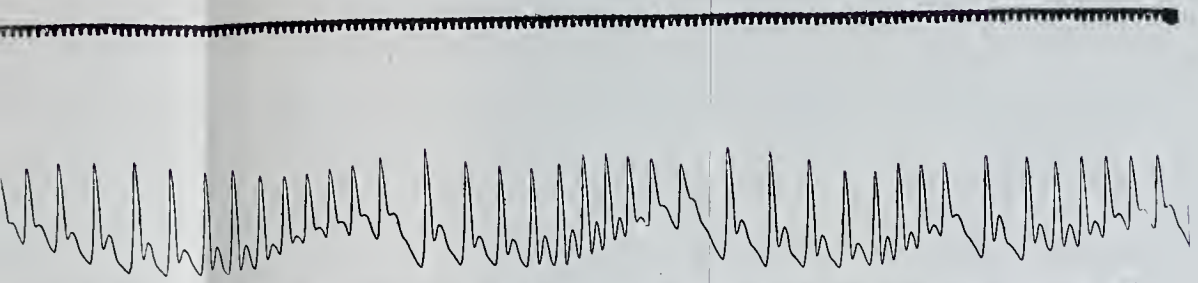
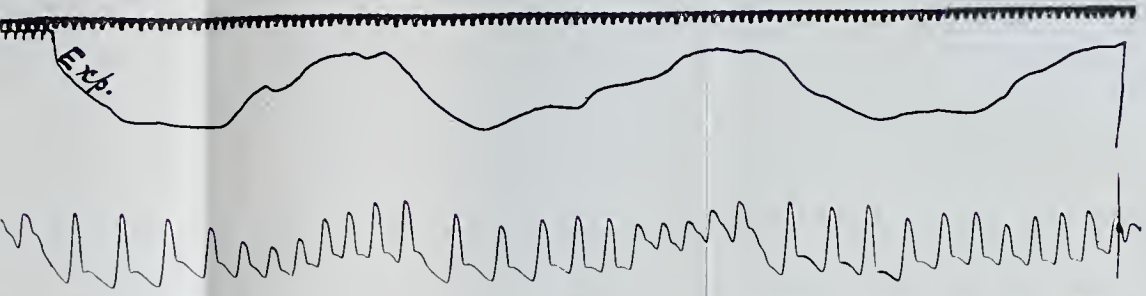


Fig. 25. Youthful type of irregularity. Healthy boy of 7 years. Radial tracing shows varying lengths in diastolic periods. The venous (upper) tracing is normal. The larger curves here are respiratory and the commencement of each inspiration is marked thus (I). Each inspiration is marked by an acceleration of the pulse (short diastole), while on expiration the pulse slows down (long diastole) as shown in the radial tracing.

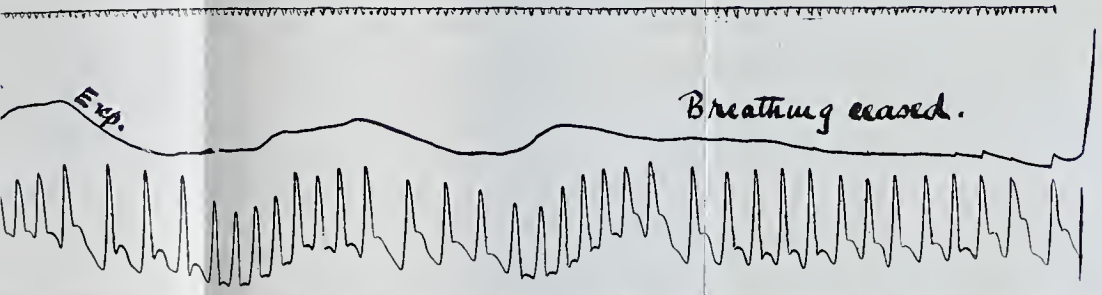
during inspiration and becomes fuller during expiration. On taking tracings I have often found this variation in the force of the beats well shown, and looked on it as rather an instrumental accident than a clinical condition. But I have also found the condition present clinically and well marked when there was no evidence of other heart disturbance. This is shown in Fig. 26, where there is a run of small beats followed by a succession of stronger and



nd irregularity. Youthful type. Healthy boy of 12 years. Clinically the pulse was found to be irregular, not the rate but also the force of the beats. The pulse tracing shows both these conditions well. Pulse rate = 85.



nd irregularity. Youthful type. Same case as above. When a respiratory tracing (upper) is taken, the effect of ulse is evident, the quick small beats occurring during inspiration, and the slower and stronger beats during te = 85.



und irregularity. Youthful type. Same case as above. To show effect of cessation of respiration on the cardiac

slower beats. This irregularity could be equally well detected by the finger on the pulse. When the respiration was taken along with the pulse (Figs. 27, 28) it at once became evident that the variation in the force as well as in the time of the beats was distinctly respiratory in origin. It is not that the respiration directly affects the force of the beats, but the quickening of the cardiac rate during inspiration is at once accompanied by a diminution in the strength of the cardiac contraction. This is the result of the general law that the quicker the heart the weaker the beat, and that slowing of the cardiac rate is accompanied by an increase in the force of the beat. The law is a physiological one.

This form of compound irregularity is therefore due in the first place to the alterations in the rate of the cardiac contractions associated with respiration, and in the second place to the alteration in the force of the beats which follows a change in rate. If the patient holds his breath or breathes very quickly the irregularity disappears entirely. If the cardiac rate is increased from any cause, such as fever or excitement, the irregularity will vanish. Like other forms of cardiac disturbance, it is most common in the nervous type of subject, and from the cardiac point of view it is of no significance and no importance.

CHAPTER IV

RAPID ACTION OF THE HEART

The rate of the cardiac action in childhood is apt to be increased much more easily and to a much greater extent than in later years. Every doctor is acquainted with the difficulty on seeing a young patient for the first time of determining how far a rapid pulse is dependent on the medical visit and how far on disease. Excitement, exercise, fever, pain, &c., will quickly increase the rate to 120, 140, or 160. In all acute diseases we are accustomed to meet with a rapidly acting heart, and also in many wasting and debilitating diseases. But in the presence of obvious disease or disturbance elsewhere we are not inclined to regard the rapid pulse as an indication of heart disease, and it will usually be found that with the disappearance of the exciting cause the heart rate quickly subsides to normal. It is different, however, when we are confronted with a case in which the rapid action of the heart is the one outstanding feature, without any obvious signs of disease to account for it. Here it is sometimes assumed without reason that the rapid pulse indicates heart disease, and this mistake may be based on the fact that a rapid pulse is common in many forms of carditis. It is therefore very important to determine whether a functional disturbance of the heart in the shape of rapid action may occur quite apart from any organic disease of that organ.

Case V. A girl, eleven years old, was admitted to hospital with the diagnosis of rheumatism and chorea, but no definite evidence of either of those conditions could be made out. During a stay of a few weeks the pulse was always rapid. On successive days it was

116—128—124—132—120—132.

She was a bright, nervous child with a somewhat anxious expression of face. There was no real change produced in the cardiac rate under anti-rheumatic treatment, although when she was resting the pulse was rather slower. Towards the end of her stay the pulse was slower, being on successive days

120—116—96—100—108.

Apart from the cardiac rate there was nothing abnormal detected on physical examination of the heart.

She was readmitted a year later with a history of the following symptoms: (1) breathlessness on going upstairs; (2) easily tired; (3) palpitation at times; and (4) frequent headaches and occasional epistaxis. In the out-patient room the cardiac rate was 170 per minute, with a tick-tack action. During three weeks in hospital there was a diurnal range of temperature of from 1 to 1½ degrees. The pulse rate when the patient was in bed varied from 112 to 124. No change followed from full doses of salicylate of soda (one dram daily). The state of the blood was normal. The thyroid gland was slightly enlarged. The gums, teeth, and tonsils were

healthy. On examination the heart was not dilated, the action was regular, there was no murmur, and the sounds at the apex were about equal in intensity. Tracings of pulse and jugular showed that the tachycardia was a sinus one. On deep slow respiration inspiratory quickening and expiratory slowing of the heart rato was present.

Observation 1. In bed, pulse rate 120. After walking exercise it ran up quickly to 140, but slowed down again after a short rest.

Observation 2. Sitting quietly, pulse rato 116. After a run up and down tho ward, pulso 138; after a two minutes' run, pulse 144.

But no other symptoms followed active exercise. She was sent to a convalescent homo for five weeks, and spent the time in the open air taking plenty of exercise. There were no symptoms of any kind, no breathlessness, and no palpitation. Sho gained 13 lb. in weight. Her temperature was normal. Tho pulse rate averaged from 94 to 84, being only occasionally between 100 and 112. When seen on her return the pulse rate was 160. There was no cardiac dilatation, and tho sounds were clear and equal.

She is now fourteen years old and is physically a well-developed and healthy-looking girl. There is no complaint of any symptoms, but the cardiac rate is usually about 120 and runs up easily to 140 per minute.

Case VI. A boy of nine years was brought to hospital because of weakness following an ulcerated sore throat. There was no evidence of diphtheritic

paralysis. General tonic treatment was given for a few weeks and it was noted that the pulse was always rapid. At successive weekly visits to the hospital it was found to be 128—130—128—124. On one visit it was 124 while standing and 146 on lying down. At the beginning of another examination it was 104 and at the end 144. The boy was described as a shy, excitable child. He was sent into hospital for further examination.

There was nothing in the boy's history suggestive of rheumatic infection. He had no symptoms of cardiac weakness or distress. The heart was normal in every respect, save as regards the rapid action. After rest in bed the heart rate quickly settled down to 90. When he was asleep it was found to be 84. At another examination when in bed the pulse rate was 86, and two hours after rising it was 128. The complete absence of any signs or symptoms in this patient led to a diagnosis of nervous tachycardia.

Observations, continued over the next six months, have shown a persistence of the conditions. Some septic and carious teeth were removed without any result as regards the heart rate. A change of posture seemed sometimes to induce an increase of rate—when the patient lay down.

Standing, pulse rate 124; at another time, 124.
Lying down, ,, ,, 146; ,, ,, ,, 140.

The boy's general health was quite satisfactory.

I think it will be found that this type of nervous tachycardia or irritable heart is not uncommon in

young subjects, quite apart from any cardiac disease. There are certain distinguishing features which must be carefully looked to. The rapid action is not an occasional but a persistent symptom, lasting for months or years, when the patient is examined under ordinary conditions. The abnormal rate would appear to have become for the time being the standard, and this rate may still further be increased by the usual excitants, emotion, exercise, fever, &c. Thus, at the doctor's visit the heart may be beating at 140 to 160 per minute, and after a little slow down to 120, but not to normal. The influence of the recumbent posture in slowing the rate is but slightly marked and may make a difference of only a few beats. On the other hand, on lying down the pulse rate is sometimes found to be increased. During sleep the pulse rate is usually much lowered and may be normal. This is an important test which should always be applied if possible. The affection known as paroxysmal tachycardia may be at times a very continuous action, but in it there is always some disorder of the cardiac rhythm present. In the type under consideration the rhythm is always undisturbed and each impulse to contraction proceeds from the sino-auricular node. The period of life when this rapid action appears is usually at or about puberty, and the subjects of it will be found to present other manifestations of a neurotic temperament.

The condition naturally calls for a careful examination of the whole system. It is well to bear in mind

the possibility of Graves's disease, even during childhood, as it is sometimes met with. As usual various sources of peripheral irritation may come under suspicion, and teeth, tonsils, and ears have been carefully looked to and treated if necessary in my cases, without any effect. Mackenzie has emphasized the importance of latent toxæmia in the production of 'poisoned hearts' with a rapid action and no other manifest change. I have carefully looked for any hidden source of infection in my cases and have failed to find it. From the fact that these patients have been under observation for years and have grown well, and have not manifested any symptoms of disturbance apart from the nervous system, I do not think there has been any latent toxæmia. The toxin which produces no disease and no marked symptoms save a rapid heart has not yet been recognized, while the subjects of 'poisoned hearts' show other symptoms of ill health, and also the effects of the cardiac disorder.

In the absence of other evidence I assume the cause of the rapid cardiac action to be a nervous one. We have at present no means of determining whether this action takes place through a weakening of the vagus control, or a stimulation of the accelerator nerves of the sympathetic system, but when one considers the accompanying phenomena in these subjects, the latter would appear to be the more likely.

The treatment should be directed to the nervous system and not to the heart.

CHAPTER V

SLOW ACTION OF THE HEART

We occasionally meet with cases in which an abnormally slow action of the heart attracts notice. This may be due to auto-intoxication, as in jaundice, or to toxins, as in influenza. In such cases we recognize the slowing as a symptom of some definite affection and are not apt to refer it to any disease of the heart.

When a persistent slowing of the heart is observed in early life, without any obvious cause, suspicion may be aroused as to the existence of heart disease. There are certain forms of slow pulse associated with cardiac disease, such as that of heart-block, which will be referred to later. The cases we are now dealing with are those in which the slowing affects all the chambers of the heart, and each cardiac contraction begins at the normal starting-place, the sinus venosus. The explanation of such a condition may at times be puzzling, as in the following case.

Case VII. A boy of eleven years was admitted to hospital suffering from a mild and first attack of chorea, which had been preceded by slight pains in the joints. The heart was not dilated, both sounds at the apex were rather feeble, and there was a soft apical murmur, systolic in time, which disappeared on firm pressure with the stethoscope. It was not

conducted into the axilla. The temperature showed a diurnal variation of 2°, rising to 99° F. at night.

On admission he was given salicylate of soda, 90 grains per diem, for three days, and then it was stopped as vomiting came on. The choreic movements soon ceased entirely.

The pulse on admission was between 80 and 90 and quite regular, but after the fourth day it fell to 60, and then gradually to between 40 and 50. On consecutive days it was 48—42—48—40—60. Venous and arterial tracings showed nothing abnormal, save that the youthful type of irregularity (respiratory) was well marked. The cardiac condition remained unchanged. The slow pulse persisted for three weeks, when the temperature rose to 102° F. owing to an attack of ulcerative stomatitis. During the pyrexial period, which lasted nine days, the pulse rate averaged 90, and afterwards it fell to between 60 and 70. A week later it was steady at between 70 and 80. After six weeks at the convalescent home, during which there had been no return of the bradycardia, he was seen again. The pulse was regular at 72 per minute. The sounds at the apex of the heart were clear, there was no dilatation, and at the pulmonary area a systolic murmur was audible. There were no subjective signs of the heart disease. During the period of bradycardia full doses of tincture of belladonna and of liquor strychninæ had been given, without any effect on the pulse rate.

The exact nature of the slowing of the heart in

this case may be open to discussion. My own opinion is that it was associated with the chorea as a part of the central nervous disturbance due to rheumatic toxæmia. The important point is as to whether such a condition of bradycardia should be regarded as cardiac in origin and due to heart disease. I have mentioned the cardiac phenomena which were present, and shall have more to say about cardiac murmurs later on. My own examinations of this patient failed to reveal any definite evidence of heart disease. There is little doubt that the boy was suffering from a rheumatic infection. The history of joint pains, an outbreak of erythema while in the hospital, and the chorea render this diagnosis certain. But an attack of rheumatism does not necessarily mean that the heart must be affected, and even in the presence of such symptoms as bradycardia and murmurs, one must recognize that the heart muscles and valves may have escaped infection. It seems reasonable in this case to assume that the temporary bradycardia was associated with a disturbed action of the vagus centre and not with heart disease.

In the case of children bradycardia of moderate degree may be a more or less persistent peculiarity of the individual, unaccompanied by any other signs or symptoms of disease. As in the above case, a marked slowing of the pulse is often found in connexion with chorea, after all active movements have ceased. It also frequently occurs after a severe illness attended by pyrexia, and is then a satisfactory

sign from a prognostic point of view. As a symptom of heart disease in youth no stress is to be laid on the presence of bradycardia ; it has to be considered in relation to the other phenomena present ; and it is not advisable to regard it as cardiac in origin unless other and definite signs of heart disease are present.

CHAPTER VI

DILATATION OF THE HEART

Dilatation of the heart as clinically determined means an extension outwards of the left ventricle, as judged by the position of the apex beat, or of the right ventricle, as judged by the line of the cardiac dullness to the right of the sternum, with marked epigastric pulsation. The apex of the heart is usually formed by the left ventricle, but in the case of marked dilatation of the right ventricle the latter may form the apex by overlapping of the left. In connexion with the subject of dilatation it is important to bear in mind that if care be not taken it may be confused with displacement of the heart as a whole, as happens in the case of pleural effusion, of fibroid lung, and of other conditions. This difficulty will be avoided if the chest as a whole is examined in all supposed cases of cardiac dilatation. Palpation of the heart is much more useful in the case of young subjects than of adults, because pulsation can be felt so much more easily through the

thin chest walls. Although percussion is also to be employed I always feel doubtful about the existence of cardiac dilatation in young people which cannot be felt with the fingers. The results of radiography by means of the ordinary apparatus have appeared to me to be particularly misleading as to the question of dilatation.

The estimation of dilatation will depend on the view we take as to what the size of a normal heart should be, and within what limits its pulsations and boundaries should be confined. Anatomically this may easily be settled, but when we come to examine the living heart we find that the anatomists prove unreliable guides. It may be granted that in many cases the apex beat of a child at rest in the recumbent posture will be found in the 4th or 5th intercostal space, and inside the nipple line, and that the right border of the heart as estimated by percussion will extend to the right border of the sternum. But clinically we find that these limits are often exceeded in perfectly healthy children. The apex beat is frequently in the nipple line or outside of it to the extent of half an inch, or more, and that in the absence of any disease, cardiac or otherwise. As the result of slight exertion or excitement the apex may be found to extend outwards, and under the same physiological conditions the right side of the heart may dilate, as shown by percussion, and by marked pulsation at the ensiform cartilage.

What must be recognized is that there is a considerable variation in the size of a healthy child's

heart under the ordinary conditions of life, and that the size is determined not by any rigid anatomical limitations, but by the amount of work which the heart is called on to do. The size of the heart is further regulated by nervous control, and this factor may lead to variations in size. For these reasons I do not think that an ordinary amount of dilatation as described above is to be regarded, when taken by itself, as an indication of heart disease in children. It is to be considered along with all the accompanying conditions of the case.

Case VIII. A girl of ten years was seen because of general weakness and depression. She had been much worried over lessons and by the school teacher. It had been noticed that during the holidays she was much better. There had been no fainting or shortness of breath. A few months previously she had suffered from a cough, which was cured by the removal of tonsils and adenoids. She had never had rheumatic fever or rheumatic pains.

The child was evidently of a nervous temperament, and had a depressed look. On examination of the heart the apex was felt one and a half inches outside the nipple line in the 4th space, and the cardiac rate was 124 per minute. The action was regular, the sounds were clear, and there were no murmurs. There was a diffused pulsation over the whole præcordial region but no sign of cardiac hypertrophy. The lungs were quite healthy.

She was kept in bed for observation. On the following day the pulse rate had fallen to 80, and

kept steady at that rate while she was in bed. The apex beat was now to be felt just external to the left nipple in the 4th space. In a few days she was allowed to be up and running about. The pulse rate was found to be increased when she was up, averaging about 124. Dilatation of the heart was sometimes present and sometimes absent. It was not uncommon to find it from a half to three-quarters of an inch outside the nipple line, without any symptoms, and accompanied only by an increased cardiac rate.

She has been under observation for a year. The only treatment employed has been directed to the nervous system, which is now in a much more stable condition. The cardiac action is usually rapid, but settles down markedly after rest. Occasionally some dilatation is present, but less marked than formerly. There are no signs or symptoms of cardiac disease.

Case IX. A boy of eight years was brought to hospital because on running he complained of pain in the left side of his chest. He had also complained of pains in the left hip and about the limbs generally. The previous year the school doctor had said the heart was affected, and that he must not run about much. The mother thought the boy was shorter of breath than other children.

At the hospital the boy was particularly bright and lively, and in this respect his mother said that his demeanour was very different from what it was at home. No evidence of local pain or tenderness

could be elicited. On examination of the heart the action was regular, 90 per minute, and the sounds were clear. The apex could be felt in the 4th space three-quarters of an inch outside the nipple line, and percussion confirmed this. He was ordered salicylate of soda in five-grain doses thrice daily.

Three weeks later his heart was normal in size, the apex being in the nipple line, the sounds were clear, and the action was regular, 88 per minute. He was made to run round the out-patient room, and the apex was then found to be half an inch outside the nipple line. He has developed no signs of carditis, and has been living the ordinary life of childhood, but the dilatation of the left ventricle is still very easily induced.

Under many forms of acute illness the heart will be found to dilate. In any fever with a prolonged high temperature dilatation may take place as the result of the fever. Under the ordinary conditions of life one feature of this physiological dilatation is its changing character, present one day and gone the next. In the case of neurotic and neurasthenic children dilatation is very common and is often more persistent. It is usually accompanied by a quickening of the cardiac action, and by a diffused pulsation over the whole præcordium which is very characteristic. Systolic murmurs may also be present. The condition of dilatation and the accompanying signs are here due in all probability to an atonic condition of the heart muscle, the result of impaired nervous control. There may be, and often is, cardiac

weakness associated, as shown on making any special exertion, but there is not cardiac disease. Such patients do not tend in any special way to develop heart disease. The treatment of the underlying nervous condition will be referred to later, and as regards the heart the presence of dilatation is no indication for the employment of cardiac tonics of the digitalis group.

Pure dilatation of the heart of functional origin must be carefully distinguished from hypertrophy. The latter is a permanent condition, the result of disease in or about the heart. The accompanying throb or heave of a hypertrophied heart is a very different thing from the flapping pulsation felt over a heart which is simply dilated, and will render assistance in the differentiation of the two conditions.

CHAPTER VII

MURMURS ABOUT THE HEART

The relation between murmurs heard in the præcordial region and heart disease is still often regarded very much as the relation between albuminuria and renal disease used to be. It was known that albuminuria was frequently associated with serious renal disease, and so it came to be assumed that the presence of albumin in the urine signified grave organic changes. Similarly, it was found that cardiac murmurs were often associated with serious heart

disease, and so it came to be assumed that the presence of a murmur signified grave heart mischief. Many a healthy boy has been made a confirmed invalid and a useless member of society by the accidental discovery of albumin in the urine, when the fact was really of no importance. Many a healthy child has been regarded as doomed to an early grave unless the severest restrictions were carried out owing to the accidental discovery of a cardiac murmur, which was of no significance. The presence of a murmur is nothing in itself. The all-important question is, does the murmur signify that the heart is or will be affected as regards its working power? This can only be satisfactorily decided by one who is acquainted with, first of all, the so-called functional murmurs of early life.

The organic murmurs of childhood are the congenital, the acquired valvular, and the acquired pericardial. With these we are not now concerned except in so far as they have to be distinguished from the non-organic murmurs. The latter are variously described under the terms 'functional', 'hæmic', 'accidental', 'accessory', 'extra-cardiac', 'cardio-pulmonary', &c. All are agreed that these non-organic murmurs are met with in early life, but opinions differ as to their frequency. Professor Rudolf found functional heart murmurs present in 60 per cent. of the inmates of the surgical wards of the Sick Children's Hospital in Toronto. I should agree that they are very common. Perhaps with the elaboration of and additions to the sensitive

properties of stethoscopes in the present day, the number of audible murmurs tends to be increased. It is a good plan to seek confirmation of the existence of a doubtful murmur by means of the plain wooden stethoscope. The audibility of a murmur varies a good deal with the observer, and a very faint murmur taken by itself is more a matter of interest to the physician than of importance to the patient.

Certain forms of functional or non-organic murmurs fall to be considered.

A very common murmur during the first two decades of life is the **pulmonary systolic.** This is so called because the site of maximum intensity is in the pulmonary area, i.e. between the 2nd and 3rd costal cartilages on the left side and close to the sternum. The murmur is a cardiac one, and has nothing to do with the lungs. From their frequency in healthy children one might almost go the length of saying that an accentuated second sound and a systolic murmur in this area are physiological in early life. The murmur may be confined to the pulmonary region, or may extend from one to two inches to the left, but as a rule it is not well conducted. In very well marked cases it may be heard as far as the apex. It often disappears during puberty, but may persist to adult life. Opinions differ as regards the ætiology of this murmur, but in all probability it is produced at or about the pulmonary valve, as the blood passes from the larger right ventricle into the smaller pulmonary artery.

The pulmonary systolic murmur has to be dis-

tinguished from the murmur of congenital defect often heard about the base of the heart. In the absence of any symptoms of the latter suggesting the origin, the diagnosis is helped by the fact that the congenital murmur is usually rougher and more rasping and of longer duration than the pulmonary systolic. Again, in the case of pulmonary stenosis there is usually dilatation of the right ventricle and weakening of the second pulmonic sound, signs which are not present in connexion with the pulmonary systolic murmur.

This murmur is not accompanied by any symptoms of heart trouble. It may be so audible as to attract attention in a marked way, but to any one familiar with it the characters are easily distinguished. It is always of a soft and blowing character, following immediately on the first sound, and short. It is not associated with any disease, local or general, and it is not of any significance from the point of view of the patient's future health.

Another murmur not infrequently heard in early life is the extra-cardiac murmur produced by the friction between the heart and lung or other surrounding tissue. That this murmur is often definitely cardio-pulmonary in origin is shown by its variation during respiratory movements. It becomes intensified on inspiration, diminishes during expiration, and disappears entirely when the breath is held. Another test for these extra-cardiac murmurs is by means of firm pressure with the stethoscope on the chest wall, when the murmur often vanishes

or is reduced in its loudness. I am aware that this is denied by various authorities, but it has proved true in my own experience. The test is of special value in the case of a child owing to the softness of the chest wall, which allows of more direct pressure about the apical region of the heart than is possible in the case of the rigid chest in an adult. The cardio-pulmonary murmur is systolic in time, and but rarely is there a whiffing sound during diastole. It is soft and superficial in character.

The murmur may vary with the position of the patient and with the rate of the heart, i.e. whether fast or slow, but the chief diagnostic test is always the respiratory one, and more especially its complete or almost complete disappearance when the breath is held. The murmur is usually heard in the area between the apex and the ensiform, i.e. the region in which the movements of the heart are freer and greater than elsewhere. If for any reason the adjoining lung tissue is encroached on by the heart it may be heard in other regions, e.g. about the base, as in the following case.

Case X. A girl of fifteen years was sent into hospital because of pain about the heart, worse when lying down. She had also suffered from palpitation and shortness of breath on exertion. The extremities were often blue and cold, and she had a tendency to cyanosis after exercise. On admission she was seen to be a healthy-looking, well-nourished girl. Functional aphonia was present, but soon passed off. There was a loud rasping systolic murmur

heard loudest at the base of the heart and to the right of the sternum. The murmur was also well heard in the neck and across the upper half of the chest posteriorly. It was regarded as congenital in origin. There was no dilatation of the left side of the heart. The right side was enlarged, there was marked pulsation at the base of the heart over the pulmonary area, and also at the ensiform. The liver was definitely enlarged. There was a triple rhythm present along the left border of the sternum. These signs were held to point to a congenital lesion of the heart.

A further double murmur was heard to the right of the upper third of the sternum and for about two inches beyond it. It was distinctly systolic and diastolic in time, very soft and very superficial. It varied distinctly with respiration, being increased during inspiration and diminished during expiration, and disappearing altogether when the breath was held. This was clearly a cardio-pulmonary phenomenon, and its appearance in this unusual situation at the base was considered to be due to some dilatation and overaction of the right heart and possibly of the larger vessels, leading to pressure on the right lung.

A definite diastolic element, as in the above case, is not common. Sometimes the interruption of respiratory sounds on inspiration has been described as cog-wheel breathing, but in the latter there is no murmur or whiff such as is present in the condition under consideration. A cardio-pulmonary murmur may be discovered accidentally in the routine

examination of a healthy heart, or it may be found in association with cardiac disease, congenital or acquired. In no connexion is it to be regarded as anything more than an interesting stethoscopic phenomenon. It produces no symptoms, does not affect the patient or his heart, and is of no prognostic significance.

Intra-cardiac and presumably **valvular murmurs** may arise without the existence of any organic lesion. It is necessary to remember that the heart is an organ formed to regulate the blood-supply to the tissues, and with a self-protective mechanism to regulate any overstrain. The valves are not only a means of directing the onward current of the blood by preventing a reflux, but also serve to relieve any over-distension of a ventricle by allowing a certain amount of reflux. This is the safety valve action of the cardiac mechanism, and we know how easily and often such a reflux takes place at the tricuspid valve, which even in the normally acting heart is barely sufficient to close the orifice (John Hunter). In certain forms of debility of the heart muscle this reflux may take place at the mitral orifice, and its occurrence may be manifested by a systolic murmur which is usually referred to the mitral valve. Probably in many cases a reflux takes place without the production of any audible murmur. In other cases the reflux takes place normally on active exertion, and some children will invariably be found to develop a murmur, usually in the mitral or tricuspid area, after active exercise, and disappearing

after rest. The production of a murmur under such conditions is generally recognized as of physiological rather than of pathological interest. It is also well known that systolic murmurs usually regarded as functional may be present about the heart during the course of, or for some time after, any acute debilitating illness, such as a specific fever, and they do not as a rule excite any fear of cardiac disease. They disappear during convalescence.

It is different, however, when a murmur of this character, apparently valvular in origin, is discovered accidentally in a young person not suspected of heart disease. Too readily sometimes the assumption is made that the murmur is evidence of heart disease. In nine cases out of ten the suspected murmur will be a mitral systolic. The patient may be described as being 'run down', 'weak', 'always tired', 'anæmic', or some such term suggesting general debility. If the patient is really anæmic that condition may serve with some as an explanation of the murmur, but there is no evidence that any alteration in the constituents of the blood is sufficient to produce a mitral murmur. Children are often anæmic and suffer from profound forms of anæmia, but in none of these are cardiac murmurs specially marked. On the other hand, the debility associated with anæmia may be a factor in the production of a murmur. Amongst the subjects of these cardiac murmurs a condition of general debility is usually present, and the most common factor in the production of this state is a disturbed and

enfeebled condition of the central nervous system. The signs of debility are manifested in other organs besides the heart, and it is only on the common basis of the nervous system that one can correlate them. The neurotic and neurasthenic young person is the typical subject for the development of functional murmurs. I am not suggesting that a functional cardiac murmur is a sign of nervous disease or disorder, but only that the resulting debility may be shown as regards the heart in the production of a murmur. When a condition of general debility is present, and is shared in by the heart, the same tendency to murmurs may be present, whatever the original cause may be.

There are many explanations given of the disturbance of the cardiac mechanism leading to the production of such murmurs, which are admittedly valvular, and usually mitral. The explanation which appears to be the most convincing and the most generally applicable is that given by Mackenzie, who emphasizes the importance of a loss of tonicity in the cardiac muscle. The ventricular wall may have become impaired in its tonicity generally, or there may have been a special weakness in the papillary muscles. The result is that the valve segments do not close with their normal promptness and completeness at the beginning of systole, and a reflux takes place. Dilatation of the ventricle may be present, an additional evidence of muscular atony, but it is not a necessary accompaniment. The cause of the murmur lies not in any defect or disease of

the valve but in the weakness of the myocardium of the ventricle.

The differentiation of these functional valvular murmurs may be made in some cases from a study of the murmur itself, but it is always advisable to take into account the presence or absence of constitutional or local disease, and more especially of rheumatic infection. We shall suppose that the sole evidence of cardiac disturbance is a cardiac valvular murmur, with or without some dilatation of the left ventricle.

Such a murmur, if functional, is always systolic in time. The sounds of the heart may be altered as regards their tone, but they are never absent. As a rule the soft and short character of the murmur is in marked contrast to the louder and more prolonged murmur of organic valvular disease of some standing. Functional murmurs vary more from time to time, even under similar conditions, than the organic murmurs. It is not at all uncommon to find them present at one time and entirely absent at another, so that the term 'fleeting' often suits them very well. The murmur is not usually well conducted, and speaking generally its area of distribution will be confined to the præcordium, with a site of maximum intensity over one or other of the valvular areas. The marked changes which take place in varying conditions, such as lying down, sitting up, and after exercise, cannot always be accurately forecasted, but should be tested in all cases.

These functional murmurs are to be distinguished from those of congenital origin. When the evidences of a congenital lesion are pronounced in the form of cyanosis, dilatation of the right heart, clubbing of the fingers, and a loud rasping murmur, the diagnosis is not difficult. When a murmur is the only evidence the differential diagnosis may be more difficult, as I have found by experience. A soft systolic murmur may be heard to the left of the sternum, not affected by respiration or by pressure or by change of position. The question may arise, is the murmur due to a slight leakage at a valve or to some cross-current through a patent interventricular septum? For practical purposes the problem is not of great importance in the absence of other signs and symptoms of heart disease. If the murmur is an isolated phenomenon it may be left alone and the patient may be allowed to pursue an ordinary life. These functional murmurs have also to be distinguished from those due to acquired disease, and this subject will be considered in a later section.

Taking the functional murmurs as a whole, the first thing the young practitioner has to do is to recognize the fact that they exist. He cannot start with a knowledge acquired by experience of the mysteries of cardiac murmurs. He can, however, begin his work with the knowledge that functional murmurs are common in youthful hearts, and that a murmur does not necessarily imply heart disease, and that a murmur should not be treated like a hothouse plant. He will then be able to learn the

characteristics of functional murmurs for himself, and to pay attention to all the other signs of disease which are present. As the term 'functional murmur' has been used as indefinitely as the term 'irregular heart', I have tried to analyse in both conditions the chief types met with, and to give their distinguishing features. My conclusion is that in early life and all through adolescence these three types of functional murmurs are common and should be recognized:

1. **The pulmonary systolic murmur**—so common that it may be reckoned as physiological.

2. **The cardio-pulmonary murmur**—a stethoscopic phenomenon of no importance.

3. **The systolic murmur of cardiac debility**—produced at a valve, but not of itself evidence of cardiac disease.

CHAPTER VIII

SUBJECTIVE PHENOMENA OF CARDIAC DISTURBANCE

THERE are certain subjective symptoms which suggest to the lay mind that heart disease is present and so medical advice is sought. Thus, breathlessness on exertion, palpitation, fainting and faintness, giddiness with pallor or flushing, and præcordial distress are known to be frequently associated with serious cardiac disease, and the presence of one or other comes to be looked on with anxiety. It is therefore essential for a thorough appreciation of

the value of these symptoms to recognize that one or all of them may be present in early life without any disease of the heart. We may go further and say that in the vast majority of cases they are unaccompanied by any disease of the heart, and are referable to some other organ or organs.

Instead of dealing with these subjective phenomena separately, I propose to refer to a larger class of young patients in whom these symptoms are especially common and especially well marked. I mean the neurasthenic patient. Mackenzie objects to the term 'neurasthenia' in this connexion, but is familiar with the condition present, which he calls the X-disease. He prefers to show his ignorance of the exact nature of the affection by a non-committal title, rather than cloak it by what he considers the meaningless term of 'neurasthenia'. The term neurasthenia, however, is used only to the predominant symptom of the affection and not to specify a disease.

The subjects of this affection are often brought for advice because of suspected heart disease, owing to the manifestation by them of some of the subjective phenomena mentioned above. A medical examination will usually reveal some of the cardiac disturbances which have been described, e.g. tachycardia, dilatation, murmurs, &c. Fainting and faintness are frequently the leading symptoms. Now fainting is by no means a common symptom in connexion with heart disease in early life. The usual history is that the patient fainted on rising in the morning or after standing for some time. The

fainting is really associated with the adoption of the erect posture, and the explanation is that the heart is not able to adapt itself quickly to the altered conditions of the circulation on the change of position. The tone of the arteries is low, blood accumulates in the large veins, and more especially in those of the abdomen, and with a defective supply of arterial blood to the brain the patient faints. Marked pallor of the face may be present during an attack, and complete loss of consciousness. The quick response to the altered conditions of the circulation under change of position or moderate strain which is automatic in the case of a healthy and normal child is wanting in these patients owing to the atonic condition of the heart and blood-vessels generally. For the same reason breathlessness and palpitation are easily excited under but slight provocation, and in some patients præcordial distress may be complained of.

Confirmatory signs of the underlying neurasthenia will usually be found on examination. The child or youth—for the condition usually arises during adolescence—complains of tiredness, becomes dull and apathetic, cannot keep his attention fixed at school, cannot remember his lessons, and becomes upset in consequence. The control of the emotional centres is in abeyance and crying fits are common. The tired and worried expression of the face becomes fixed. Evidence of a general atonic condition is present. The pulse is specially likely to attract attention because of its extremely low tension and

small size, so that if the hand be held up above the head the pulse seems to disappear almost entirely. In marked contrast apparently there is a large area of cardiac pulsation, and the heart can be felt throbbing violently in a manner which at first suggests cardiac hypertrophy. It will be found, however, that this is merely a somewhat excited action of the heart, simulating overaction, but without any real force, and that the weak pulse corresponds directly with the weak cardiac action. Both of the heart sounds are usually weak, and the second at the apex is often as loud as the first. A rapid action of the heart, 110 to 120 beats per minute, will often be present, or is very easily induced by any exertion. Dilatation of the heart may be found, the apex beat being situated half an inch or more outside the nipple line. Cardiac murmurs may be present of the type already described in connexion with dilatation and atony of the heart muscle. Peripheral stasis of the circulation is manifested by cold and dusky hands and feet, and by a chronic flush on the forehead, the cheeks, and the chin. Albuminuria is often present of the orthostatic variety, i.e. the albumin appears only after the patient has been standing or walking, and is unaccompanied by any other signs of organic disease. The stomach and bowels are atonic and dilated, and constipation is usually a marked feature. Headaches, backaches, and pains about the præcordium and the splenic region are often complained of. In addition to the chronic condition nerve storms arise

occasionally in the form of prostrating attacks of sickness and headache, sleeplessness, and moderate delirium.

It will thus be seen that manifestations of the so-called ' weak heart ' may be present in abundance, but in this class of patient the real evidences of heart disease are absent. The symptoms of an excitable and exhausted nervous system are usually well marked. Throughout this section it has been argued that cardiac disturbances occur quite apart from any disease of the heart. The existence of a large class of patients, the neurasthenic, in whom these disturbances are manifested in great variety and whose freedom from cardiac disease has been confirmed by prolonged observation, supports this argument. In this type of patient functional disturbances of other organs are usually found if looked for, and will prove a strong confirmatory factor in the diagnosis. Supposing that some other organ has come under suspicion, e.g. the stomach from indigestion, we may find that the stomach is dilated and atonic. Splashing of a very marked character can often be elicited two or three hours after a meal. In such a case one does not diagnose disease of the stomach, but considers the condition as a functional one and dependent on outside influences, to which treatment should be directed. So it is with the manifestations of the ' weak heart ' referred to. They are not connected with any inherent disease or debility of the heart, but are dependent on outside influences, to which treatment should be directed.

It is during adolescence that this type of neurasthenia usually appears, and in all probability the developmental changes through the system have a good deal to do with it. Similar subjective symptoms, which appear to depend on circulatory disturbance, may appear under other conditions, for instance any severe or prolonged illness. They are often seen after an attack of influenza. They may appear in young people who are run down from any cause. They may be brought about or aggravated by any excessive physical strain or marked excitement. In short, these subjective symptoms, although met with most typically in neurasthenic patients, may be found under any conditions which tend to exhaust the system generally.

It is especially important to recognize the existence of these subjective phenomena as a thing quite apart from heart disease. Their significance in early life is entirely different from what it is in later years. In adults the presence of breathlessness, faintness, &c., is often suggestive of organic heart disease, which may be latent so far as physical signs go. In early life, on the other hand, they are not *per se* suggestive of heart disease. In early life we have this further test, which in my experience is extremely reliable, that if the subjective phenomena are due to heart disease there will be marked physical signs of carditis or of a pathological rhythm. In the absence of the physical signs of heart disease we are not justified in ascribing merely subjective phenomena to such a cause.

CHAPTER IX

PROGNOSIS AND TREATMENT

We have already considered the prognosis in connexion with some of the cardiac disturbances, and pointed out that many of them are of no importance, and of no prognostic significance. A prognosis may be stated in precise terms, or it may be hinted at by the use of some non-committal term. In connexion with any suspected affection of the heart a loosely applied term may convey a very grave significance to the lay mind. If a child is stated to have 'a weak heart', or 'a murmur about the heart', or a 'dilated heart', or 'something wrong with the heart', that child is at once looked on by the parents as being the subject of heart disease. We may not be asked for any other prognosis, or we may give a reassuring prognosis—it matters not, for the lay mind assesses at its own value the presence of heart disease. Hence it is advisable in connexion with these cardiac disturbances not to use any terms which could possibly suggest heart disease.

From the medical point of view the prognosis will depend on what view the physician takes of the significance of the disturbance. The whole of this section has been on disturbances which I regard as quite apart from heart disease in a medical sense. Consequently the prognosis does not depend on the

cardiac disturbance but on any underlying disease or disorder of some other part of the body which is the primary cause of the disordered action.

The question of treatment is linked closely with that of prognosis. When these irregularities and murmurs and other forms of disturbance were insufficiently understood, it was too often the custom to anticipate cardiac disease or debility in the future, and to adopt more or less restrictive methods of treatment. The young person was restricted in all his habits and exercises, and often made a confirmed invalid because of suspected heart disease, and of possible ills that might follow. If nothing did develop this happy result was put down to the restrictive measures adopted. If the disturbance persisted the restrictions were rigidly persevered with. With a clearer knowledge of the meaning and significance of these disturbances such lines of treatment are now considered not only useless but harmful.

It may not be possible to pronounce at once as to the significance of every form of disturbance. Regarding the youthful type of irregularity, one may state definitely and at once that it is of no importance and calls for no treatment. Regarding a cardiac murmur, one may find it advisable to watch the case for a time, before definitely deciding as to the significance of the murmur. As already stated, a knowledge of cardiac murmurs and their significance is to be acquired only by experience. A definite decision must be come to as to whether

the case is one of cardiac disturbance or of cardiac disease before any treatment is adopted.

When it has been decided that the case is one of cardiac disturbance as described in the preceding pages, the important conclusion that follows is that no treatment directed to the heart is required. The patient may require to be treated, and the following suggestions are made as to the line of procedure :

1. Consider the patient, and his surroundings, and his mode of life. Often it will be found that there will be some source of unrest in these conditions which has put the system out of tune, and the removal of which leads to the disappearance of the cardiac disturbance.

2. Search for any source of irritation or any disease in other parts of the body. In many cases the cardiac disturbance is secondary to disease elsewhere, or possibly to gastro-intestinal disorder.

3. Examine very carefully into the state of the nervous system. I believe that the majority of the disturbances which have been considered are to be traced to a disordered state of the nerves, and will not be removed except by treatment directed to building up a healthy nervous system.

SECTION B

THE BORDERLAND BETWEEN FUNCTIONAL DISTURBANCE AND ORGANIC DISEASE OF THE HEART

CHAPTER X

PAROXYSMAL TACHYCARDIA

It is difficult to draw a hard and fast line between functional disturbance and organic disease in some cases. As we have seen, functional disturbances are usually caused by factors outside the heart acting through the nerves. Organic disease, on the other hand, is due to changes in the endocardium, myocardium, or pericardium, the result of an infection or poisoning of these tissues. Generally speaking, the distinction between these two classes of cases is not difficult to make, if not at one examination, by careful watching of the case for a short time. The problem is more difficult under the following conditions. Suppose that a heart previously healthy becomes suddenly disturbed in its action and symptoms of cardiac failure supervene. Sudden changes take place from time to time, the disturbance ceasing and the cardiac action becoming normal, with a rapid disappearance of all symptoms. We may see before

us the results of cardiac failure as usually associated with grave organic disease, while we can discover no definite signs of heart disease, but only the evidences of disturbed action. This problem is well illustrated in connexion with paroxysmal tachycardia. That symptom may be present in connexion with the organic changes of heart disease in adult life. But here it is to be considered as a disturbance arising in a previously healthy heart. It may be said that the occurrence of paroxysmal tachycardia is evidence of pre-existing disease. But if so, then it is a form of disease of which we have no knowledge. Hutchison and Parkinson have recorded a case of paroxysmal tachycardia in a child, aged $2\frac{3}{4}$ years, in whom no other signs of disease could be detected. Four attacks in all had been observed, the pulse rate being usually 245 per minute. The condition has been carefully examined and former disease, former infection, and signs of organic disease of the heart before, during, or after an attack, have been looked for and found wanting. When the heart is examined between the attacks it will present no objective signs of disease and the patient has no symptoms of cardiac distress. In the present state of our knowledge of this condition all we can say is that there is a disturbed action of the heart, which, if sufficiently prolonged, will lead to symptoms of cardiac failure. For this reason I have placed paroxysmal tachycardia, occurring in an apparently healthy heart, in the borderland division between functional and organic diseases.

Case XI. Male, 9½ years old. With the exception of nocturnal enuresis, cured by moderate doses of atropine, and croup this boy had had no previous illnesses.

The first known attack of 'heart hurry' began at the age of 7½ years. He was at the theatre when he complained of his heart 'thumping'. This was attributed to excitement, but as it persisted medical advice was sought. A serious view of his condition was taken by several doctors. The boy was kept at absolute rest in the recumbent position with ice-bags to the præcordium and head. Brandy and milk were ordered.

These measures had no effect and another doctor was called in, to whom I am indebted for the following notes: The boy looked well. There was no lividity or dyspnœa or œdema. The radial pulse was uncountable. The apex beat was outside the nipple line and the cardiac rate was over 200, so that it could not be accurately counted. The liver was enlarged and slightly tender. The boy expressed himself as feeling well. This condition remained unchanged for six days, when about three o'clock one morning the nurse in charge, while trying to count the rate of the heart-beats, felt the heart give a 'flop' and immediately the action became so slow that she felt alarmed. Soon afterwards the doctor found the heart beating at 40 per minute, with an occasional irregularity every 14 or 15 beats. The pulse remained very slow for a week, when he seemed so well that he was allowed out of bed, and as soon

as he began walking the pulse became quite regular at 80 to 90 per minute. The cardiac dilatation had passed off.

After remaining well for a month he had an attack of measles, which ran a normal course without any cardiac disturbance. At intervals of about two months attacks of tachycardia occurred, the shortest lasting ten hours, and the longest two days. When vomiting was induced by means of calomel the heart usually at once became quiet, and this had become the routine treatment. The boy took so little notice of the attacks that medical advice was not usually sought.

At the age of 9½ years an attack began which calomel failed to relieve. Belladonna, bromides, cannabis indica, chloral, and opium were all pushed to their full physiological effect without producing any change in the heart. His cardiac rate was from 180 to 200 per minute, and this persisted whether he was sleeping or waking, at rest or after free movement, on starvation or full diet. I saw him for the first time three weeks after the onset. Digitalis was given in moderate doses. No change took place until the thirty-third day of the attack, when the cardiac rate dropped to 80 and the boy was soon well again.

A few weeks later another attack began and lasted for a month, when, as signs of cardiac failure had set in, he was admitted to hospital. Apart from the cardiac condition there was no evidence of any disease in any part of the body. The face was rather

puffy, and there was some œdema of the lower extremities and the abdominal wall ; the ears were dusky in colour. The liver extended two inches below the costal margin, and there was a fluid thrill over the abdomen. The breathing was rather laboured, but there was no evidence of œdema of the lungs. The heart rate was 180 per minute, and regular. There was a diffused præcordial pulsation, not forcible, and the left border of the heart was two and a half inches to the left of the nipple line, while the right extended half an inch to the right of the sternum. There was very marked venous pulsation in the neck. The radial pulse was very small, feeble, and rapid, but apparently all the cardiac pulsations reached the wrist. He had been taking thirty drops of tincture of digitalis daily at home for a few days, and this was continued.

On the fifth day after admission diuresis set in and the dropsical signs rapidly disappeared, while the boy felt generally relieved. The evidences of marked cardiac failure did not again recur. The cardiac rate had fallen to 96, the action was much stronger, and the dilatation was less marked. Venous pulsation in the neck had ceased. The digitalis was stopped four days later but the heart rate was not continuously lowered, for at times it shot up to 170.

For nine weeks the heart continued to alternate between slow and fast periods. When he was taking digitalis the slow periods were increased in frequency and duration ; when he was not, the tachycardia

predominated. Intermittent courses of digitalis were given as follows :

Tr. Digitalis m. xxx per diem :
April 29 to May 5 (6 days).
May 11 to May 16 (5 days).
May 25 to June 8 (14 days).

The patient was always conscious of the onset and offset of the rapid rate, but its presence did not necessarily cause him any disturbance. The persistence of the rapid rate for a few days led to a feeling of tiredness and disinclination for exertion. He was often up and walking about irrespective of the rate of the heart's action.

In July he was put on digitalis again, 45 drops of the tincture daily. During nineteen days he took eleven drams in all, a dose being omitted occasionally when nausea or headache was present. This had the effect of producing marked instability of the cardiac rate so that it changed from fast to slow with great frequency, on the slightest stimulus, and usually without any recognizable cause. The patient stated that he could always change the rate by breathing deeply. Pressure on the vagus nerve on either side of the neck was not well tolerated owing to pain being elicited, so that the effect of this test could not be determined. In the slow period the heart rate would at one time be 42 and then change to the normal 80–90 beats. The arterial pressure, as estimated by the sphygmomanometer, was 100 during the slow period and 95 during an attack of tachycardia.

As the slow periods became longer the dilatation of the heart became less. When the patient was up the response to effort was singularly good, especially when one considered his condition on admission.

At the end of this course of digitalis the heart became slow for a fortnight, with very few and very short attacks of tachycardia. He was discharged and ordered to take the digitalis occasionally, 30 drops daily, if the attacks recurred. He went to the seaside and began walking about, and then took his ordinary exercises, including sea-bathing. The attacks steadily diminished in frequency, and after a few weeks occurred only once in two or three days and for a few minutes at a time.

He was seen again two months after his discharge. He was at school, and walked a mile to it from his house. The boy looked well and complained of no subjective symptoms. The cardiac apex was half an inch outside the nipple line and the transverse measurement was $4\frac{3}{4}$ inches (in hospital it had sometimes been 7 inches). With the exception of an occasional extra-systole (auricular) the cardiac action was regular and the rate was 48 per minute. At the pulmonary area the second sound was reduplicated (as had been noted at times previously), but there was no murmur anywhere and no venous pulsation in the neck. The præcordial pulsation was good, but did not suggest hypertrophy. The liver was not enlarged.

While the individual cases vary in certain features the above may be taken as a typical case.

As regards the character of the attacks we find that they begin and end abruptly. When the heart is in this excitable condition any slight disturbance will set it off. But the same influences will not act if the heart is not excitable. In this patient, as in others, vomiting for a time served to check an attack, but later it failed to act. When the heart was unstable we found that tapping the præcordium would often stop an attack or induce an attack as the case might be. The abrupt onset and subsidence of attacks serve to distinguish this condition from other forms of tachycardia due to excitement, exertion, Graves's disease, and acute illness of any kind. The start and finish are usually marked by symptoms of which the patient is conscious. Here is the boy's own description : ' When my heart goes off it flutters a great deal and then steadies down. I can tell as soon as it goes up by the jump it gives. After the fluttering has stopped I can hardly feel my heart beating. When my heart goes down there is a sharp stabbing pain in my neck and then the heart goes very slowly. My heart goes down almost at the same time as I have the pain in my throat.' During the attacks the boy was usually kept in bed at first, but expressed himself as anxious to get up as he felt quite well. Eating and sleeping were normal. When he was out of bed during the periods of rapid heart action, the only thing noticeable was that he seemed less inclined for exertion than under normal conditions. The short attacks were not accompanied by any oliguria or polyuria. In the

hospital he had no attacks of abdominal pain, but stated that at times there had been pain and tenderness in the region of the liver, and that he had had attacks of gasping for breath.

In this affection the periods of rapid heart vary from a few seconds to hours, days, or weeks. All of these variations were present in this patient. Before he came into hospital the attacks had been prolonged, and so far as could be determined by the intermittent examinations made there had been a period of four weeks during which the heart had not slowed. In all probability, however, the heart had slowed down at times, as judged by the conditions when he was in the hospital. The effect on the heart is undoubtedly proportional to the duration and frequency of the paroxysmal attacks. If there are no intervals there tends to be progressive exhaustion of the heart muscle. If there are sufficiently long intervals it is surprising to find how rapidly the heart is restored to a normal state.

There are certain points to be noted about the heart and circulation during an attack. The rate is usually from 160 to 200 per minute. In this patient it ran very constantly from 170 to 180 when estimated by graphic records. In slight and short attacks there is probably little change to be made out in the size of the heart, but if the seizure is at all prolonged dilatation is usually a marked feature. This affects both sides, but is more easily detected on the left side. The apex beat may be felt to extend two inches or more to the left, and this may

persist throughout the attack. The amount of the dilatation will usually be found to be proportional to the frequency and duration of the attacks of rapid heart. While examining the heart at the onset of an attack I have sometimes found the apex beat pass outwards, almost at once, for a distance of one inch as compared with its position during the slow period. As Dr. Thomas Lewis has pointed out, mere rapidity of action does not tend to induce cardiac dilatation but rather the opposite condition, and so we must regard the dilatation in paroxysmal tachycardia as due to some embarrassment of the left ventricle, possibly also of the right, in their action. A systolic murmur may sometimes be heard at the apex when the heart is dilated, but was by no means much in evidence in the present case. A striking feature is pulsation in the veins of the neck, which is not only visible but also palpable. Abnormal pulsation in the neck is recorded in old reports of such cases, and is usually ascribed to the pulsation of the carotids. Even without graphic records, which disprove this view, it can be seen and felt clinically that the pulsation is in the veins. The larger venous wave suggests at first sight very marked regurgitation through the tricuspid valve. On examination of the heart there is no evidence of any such regurgitation. A venous tracing throws more light on the subject. It shows a very large wave preceding the carotid wave, so large indeed that it is as much out of proportion to the normal auricular wave as the pulsation in the neck is out

of proportion to the normal venous pulsation. On careful measurement I found in the above case that undoubtedly this large wave on the venous tracing was the *a* wave, i.e. was due to the contraction of the auricle. In other cases of paroxysmal tachycardia there may be a different disturbance of the cardiac mechanism, but I am dealing here with what is the commonest type in childhood.

The question arises as to why this large venous wave should be present in a rapidly acting heart when presumably the contractions are weaker than usual. Examination of the venous tracing showed that the auricular wave occurred during the sphygmic period, i.e. while the ventricle was still in a state of contraction. The result was that the contraction of the auricles was quite ineffectual in driving the blood on through the tricuspid and mitral valves which were closed by a higher pressure on the opposite side. The blood could escape on the right side only through the sino-auricular opening, and so it was to some extent forcibly returned into the large veins, and produced the marked pulsation in the neck which has been described. It will thus be seen that the auricles, as part of the mechanism for promoting the flow of blood through the heart, had for all practical purposes been thrown out of action. Their contractions were quite regular but they came at the wrong time, so that the normal auriculo-ventricular co-ordination was lost.

We have already referred to the changes in the ventricle during attacks of moderate duration. The

patient as a rule shows no signs of distress, and the breathing is not affected. But if the cardiac condition persists continuously for weeks, or what amounts to the same thing with only brief intervals of slow action for recuperative purposes, signs of failure of the left ventricle begin to appear and may become very marked. The colour becomes dusky, the liver enlarges, œdema and ascites may become evident, the breathing becomes rapid, and signs of pulmonary engorgement may appear, while there may be a diminution in the amount of urine. These are the signs which we associate in cases of organic disease with a failing heart, i.e. failing power in the left ventricle. In this case the dilatation of the left ventricle became more marked with the onset of these signs. As there was presumably no organic disease of the ventricle, the conclusion seems justifiable that in early life and purely as the result of a disordered rhythm all the signs and symptoms of cardiac failure may result.

Graphic records taken in this case must be interpreted in accordance with the findings of modern cardiology. It has been shown by Mackenzie, Lewis, and others that in paroxysmal tachycardia there is an abnormal stimulus centre, which may be in the auricle, the junctional tissues, or the ventricle. We have to deal with the production of extra-systoles from a centre which is not occasionally in action, but which during the period of tachycardia takes entire control of the cardiac rate. In the language of Dr. Lewis, the ' pacemaker ' of the heart is shifted

for the time from the sino-auricular node at the sinus venosus to some other centre. What initiates this disturbance is not known, and considering that it may arise, as in the above case, in a heart previously healthy, the problem as to the exact ætiology is not an easy one. The new focus of stimulus production is most commonly in the auricle. In this case I had considerable difficulty in deciding as to whether the

Fig. 29. Paroxysmal tachycardia. [Boy of 10 years (case in text). Radial tracing during paroxysm. Pulse rate = 180. Note the regularity of the beats as to rate and force. Pulse is of the anacrotic type.

Fig. 30. Paroxysmal tachycardia. Brachial tracing. Pulse rate = 160. Pulse of anacrotic type.

ectopic focus of stimulation was in the auricle or the ventricle, but Dr. Lewis was able to state definitely from his electro-cardiographic records that it was supra-ventricular. The rate of contraction in one attack will usually be the same in subsequent attacks in the same individual, which suggests a repeated disturbance in the same spot. In some cases there would appear to be more than one abnormal focus in the auricle which initiates and takes control of the cardiac contractions for the time being.

During a rapid period the rule is that the pulse tracings show perfect regularity in the force and spacing of the beats (Fig. 29). In this case the pulse tracing was usually of the anacrotic type (Fig. 30). The regularity may at times be broken by periods in which the *pulsus alternans* is present (Fig. 31),

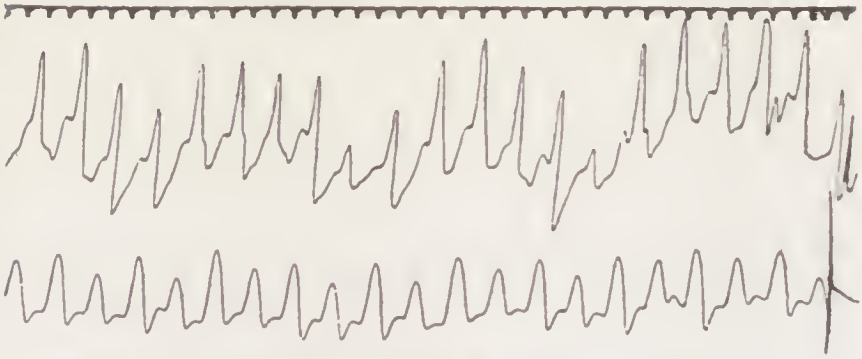


Fig. 31. Paroxysmal tachycardia. *Pulsus alternans* during a paroxysm. Pulse rate = 160.

Fig. 32. Paroxysmal tachycardia. Radial tracing during slow period. *Pulsus alternans*. Pulse rate = 55.

and I have found this also in some of the slow periods (Fig. 32). In these rapid periods the ventricular contractions are at the same rate as the auricular, and this is a distinguishing feature from some other forms of paroxysmal tachycardia in which the auricles are beating faster than the ventricles (auricular flutter). The presence of the *pulsus alternans* is also of value in diagnosis, because when associated with tachycardia it may

be taken as indicative of a disordered rhythm of the heart.

During the fast period the venous tracing is not very easily taken in all its details, and one may be misled into regarding it as of the ventricular type (Fig. 33). Repeated observations have led me to consider that in this case the auricular type of venous pulse was present, as shown in Fig. 34. The *c* and *v* waves

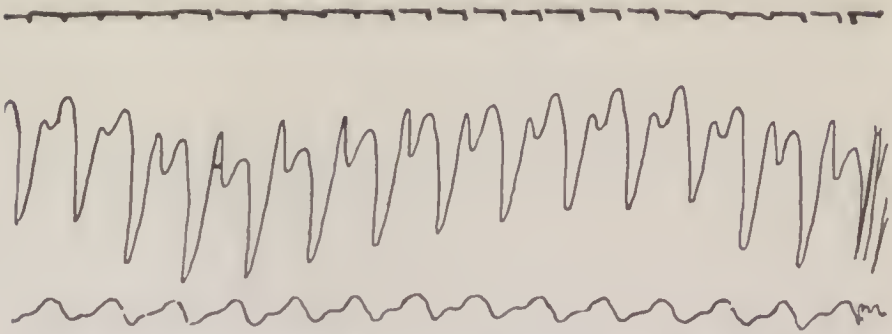


Fig. 33. Paroxysmal tachycardia, during attack. The upper (venous) tracing suggests the ventricular type of venous pulse, but careful measurements show it is not so. This tracing cannot easily be identified, but later tracings are clearer. Pulse rate = 180.

can be identified by measurement. The large wave marked *a* is clearly the result of the forcible venous pulsation seen and felt in the neck, and must be due to the auricular contraction. It occurs during the sphygmic period between *c* and *v*, and while the ventricle is still in a state of contraction. This tracing should be compared with the electro-cardiographic record (Fig. 35) which Dr. Lewis kindly took for me. In this the auricular contraction *P* is not easily identified, but apparently it occurs between

R and *T*, i.e. during the period of ventricular contraction. These tracings may be compared with one

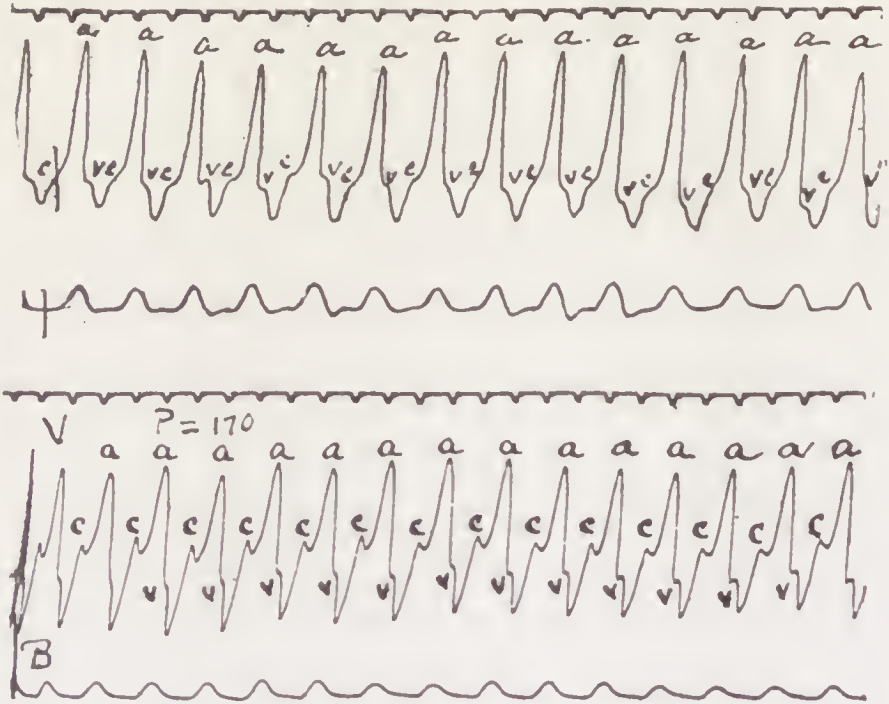


Fig. 34. Paroxysmal tachycardia. Jugulo-brachial tracings taken during a paroxysm. Pulse rate = 170. Note the regularity of venous and brachial tracings. The auricular wave (*a*) and the carotid wave (*c*) can be identified by measurement from the ordinates. The very large venous wave (*a*) is due to the fact that the auricular contractions occurred while the ventricle was still in systole and the blood was therefore thrown back into the veins.

taken between attacks, when the pulse rate was 70 (Fig. 36). The venous tracing here is normal, save that there is occasionally a slight lengthening of the *a*–*c* interval.

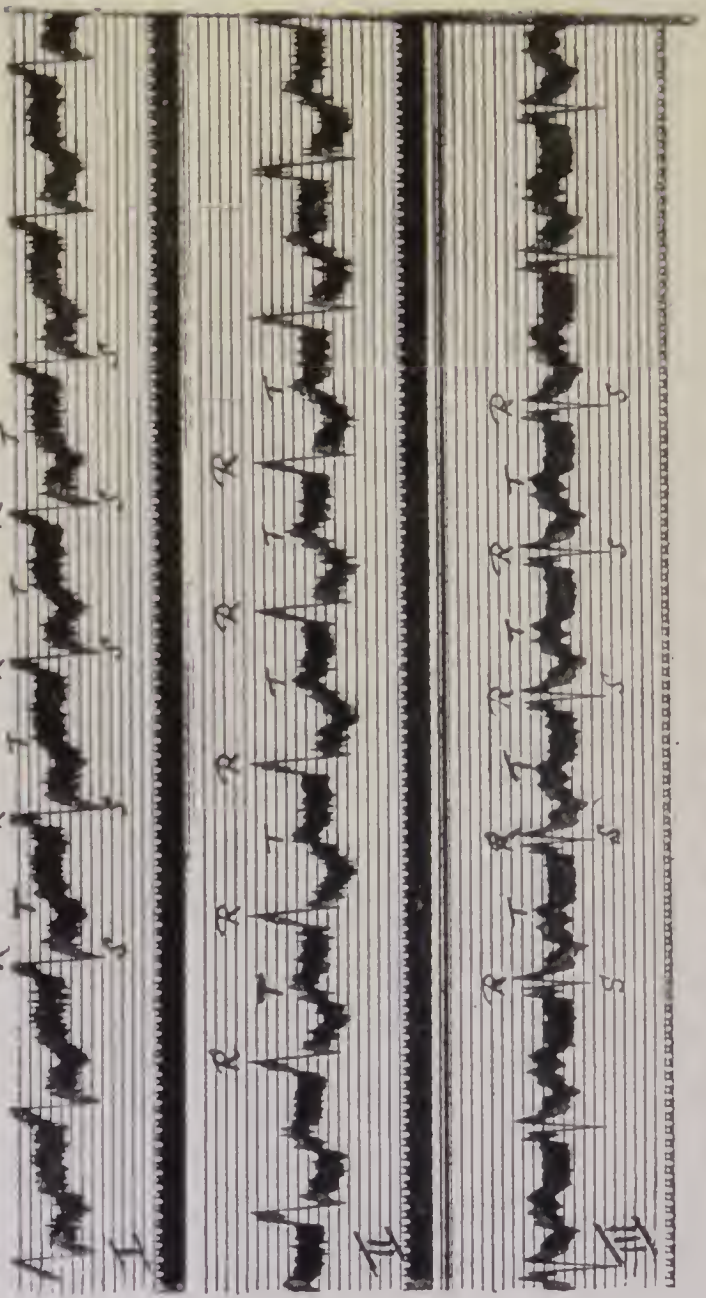


FIG. 35. Paroxysmal tachycardia. Electro-cardiogram taken during an attack.

Tracings taken of the end and the beginning of an attack, and of the intervening periods, are full of interest. As already stated, the attacks of rapid heart begin and end abruptly, and this is a characteristic feature of paroxysmal tachycardia as compared with all other conditions inducing a rapid action of the heart. Some illustrative tracings are given of the conditions referred to (Figs. 37, 38, 39).

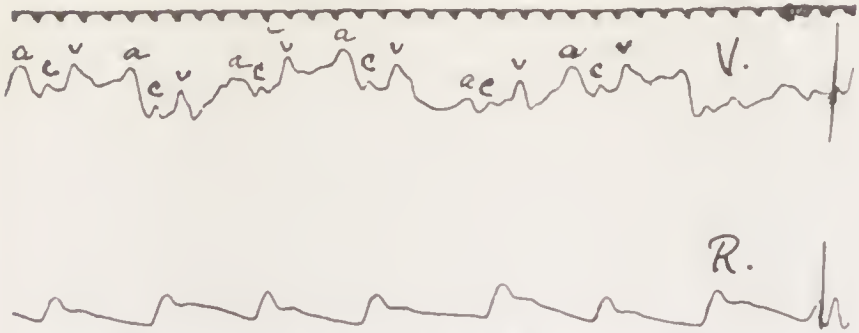


Fig. 36. Paroxysmal tachycardia. Venous and radial tracing during a slow period. Pulse rate = 70. The venous tracing is normal, save that at times the *a*–*c* interval is prolonged. Youthful type of irregularity shown in radial tracing.

The pause at the end of an attack, during which the whole heart rests, is always a long one, and may be very long as compared with the previous rate. In one of these tracings the cardiac cycle during the rapid period occupied two-fifths of a second, and the succeeding pause lasted for three and one-fifth seconds. As a rule the succeeding pauses are shorter, and gradually the normal rate and action are resumed. The period after an attack may, however, be broken by marked irregularities, both

in the form of variations in rate and force of the cardiac contractions, and in the occurrence of extra-systoles. These are usually auricular in origin, and may or may not lead to a ventricular contraction. The onset of an attack is marked by a succession of extra-systoles, which may be ineffectual at first in upsetting the old rhythm, but soon get the upper hand and establish the new rhythm. After an attack there may be a period during which the cardiac rate is unusually slow, 40 to 50 beats per minute (Fig. 40). The tracings show a sinus arrhythmia and at times what looks very like heart-block. This condition of bradycardia lasts for a time and then passes off, the normal rate of 70 to 80 being resumed. During many after-attack periods, when the patient had been taking digitalis, the slow rate leads to a manifestation of ventricular escape, i.e. the ventricle starts contracting at the same time as or even before the auricle (Fig. 41), and without waiting for the normal stimulus to reach it. In the polygraph tracing this usually leads to a large combined *a* and *c* wave. In the electro-cardiographic tracing (Fig. 42) which Dr. Lewis kindly took for me, this condition of ventricular escape is shown very beautifully. There is a sinus irregularity present, and the relative position of *P* (auricular contraction) to *R* (ventricular contraction) changes with the length of the diastolic pause. If the pause is very long the ventricle escapes from the control of the auricular rhythm.

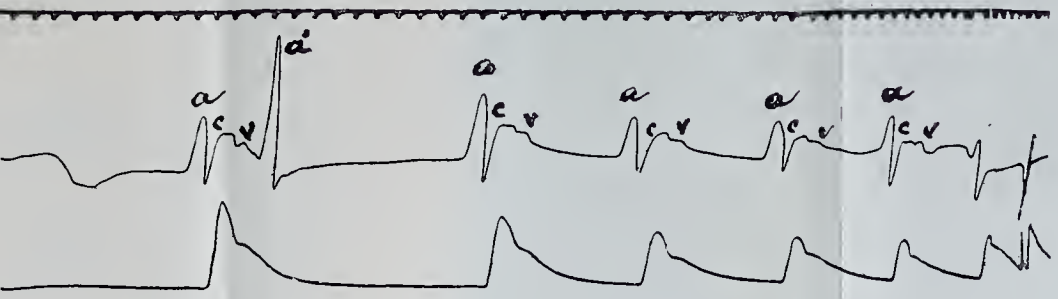


FIG. 37. Venous and radial tracing, showing the end of a paroxysm. Note the long pause in the cardiac action which follows the attack of tachycardia, and the gradual resumption of the normal cardiac action. An extra-systole(a') is shown, which is probably auricular.

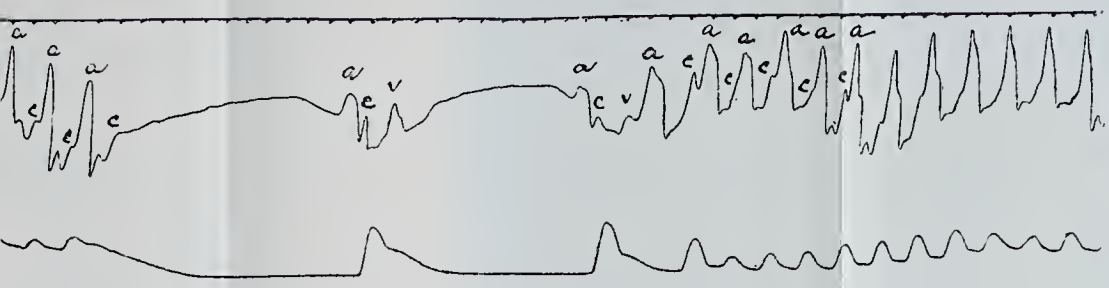
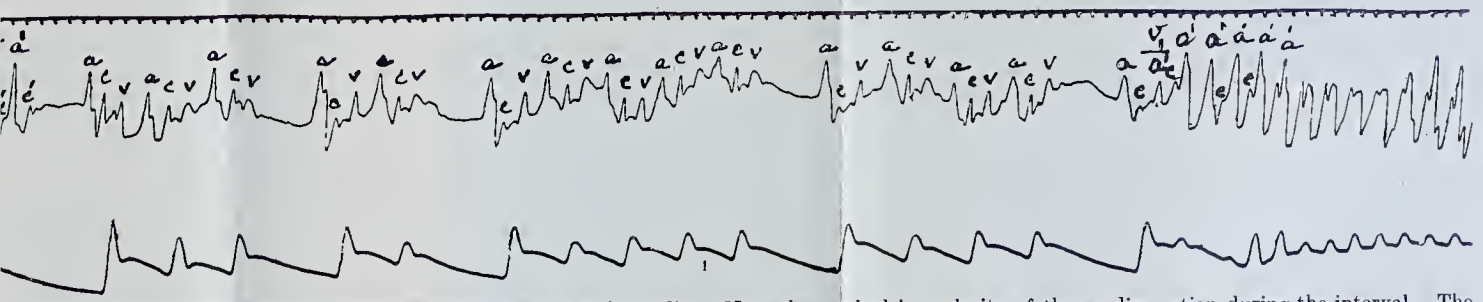


FIG. 38. Venous and radial tracing. A complete interval between two attacks is shown. The sudden cessation and also the onset of an attack are shown, the latter being more gradual.



l tracing. A complete interval between two paroxysms of tachycardia. Note the marked irregularity of the cardiac action during the interval. The regularity, and several extra-systoles which do not affect the radial pulse. The radial tracing shows a marked sinus irregularity. At the end of it the ıccession of extra-systoles, which soon gain the control of the cardiac rhythm, is well shown.

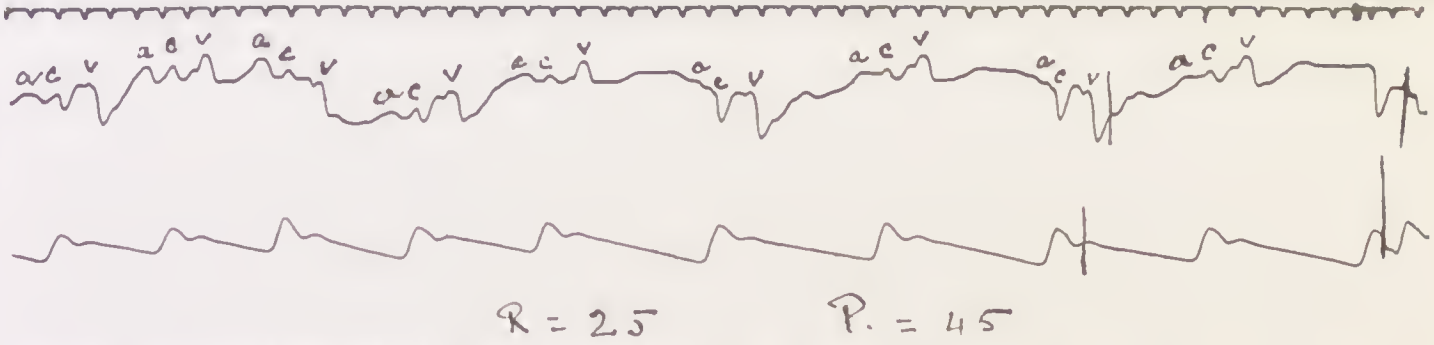


FIG. 40. Paroxysmal tachycardia. Venous and radial tracing. Taken during a slow period following an attack of tachycardia, and without any digitalis previously. Pulse rate = 45. The diastolic periods vary (sinus irregularity).

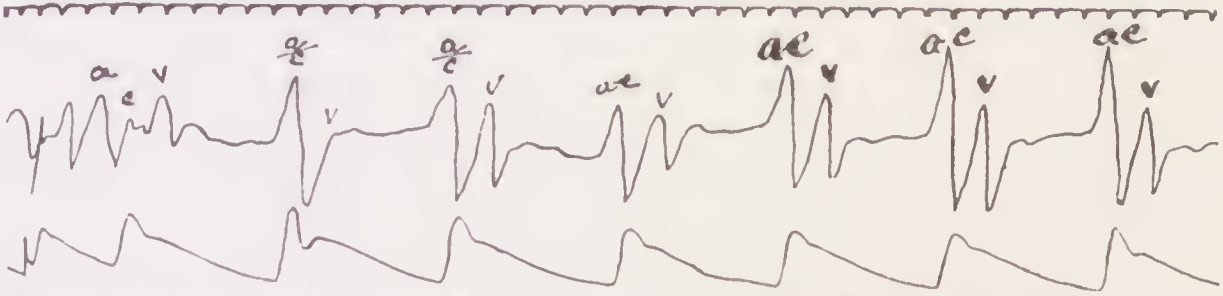


FIG. 41. Paroxysmal tachycardia. Venous and radial tracing. Taken between attacks of tachycardia. The pulse rate is slow, 50 per minute, and patient was under the influence of digitalis. Ventricular escape shown, i. e. when the diastole is long, the ventricle contracts without waiting for auricular stimulus. Here ventricle and auricle contract at the same time as shown by the large combined *a* and *c* wave. When the diastole is shortened the normal rhythm reappears.

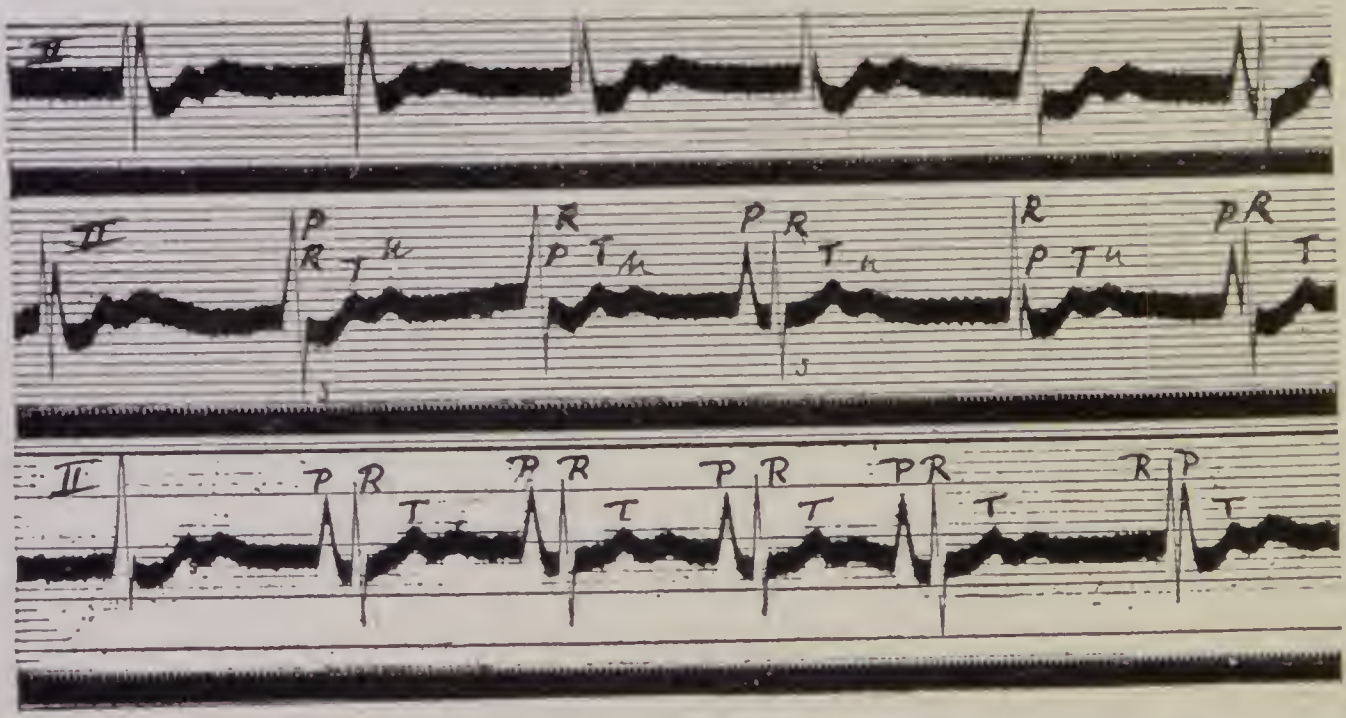


FIG. 42. Paroxysmal tachycardia. Electro-cardiographic tracing. Ventricular escape. Slow action of heart between attacks of tachycardia. The ventricular contraction (*R*) occurs sometimes after, sometimes with, and sometimes before the auricular contraction (*P*).

FIG. 43. Paroxysmal tachycardia. Venous and radial tracing. During a slow period, after patient had been taking digitalis. Two different rates of cardiac action, the faster at 75 beats and the slower at 45 beats per minute.

The study of a case such as the above leads one to view cardiac pathology in a different manner from what was formerly done. It was sufficiently simple to say that there was a disturbance of the innervation of the heart which led to the tachycardia. It is not to be denied that certain nervous disorders may lead to very rapid action of the heart, up to 200 beats per minute. This is more especially marked during childhood, and may be seen in connexion with cerebral defects and cerebral diseases. In such cases, however, there is no evidence that there is any alteration in the cardiac action save an increase of rate. In paroxysmal tachycardia, on the other hand, we have in addition a disturbance of the mechanism of the heart producing a disordered rhythm, and the site of this disturbance can now be localized in the substance of the auricle, ventricle, or junctional tissues. This fact does not of itself disprove the possibility of the disturbance being of nervous origin, but it certainly disproves the view that the main nervous tracts—vagus or sympathetic—are at fault. The intimate innervation of the heart substance is so little understood that it must be left an open question whether this special form of cardiac disturbance does or does not arise from any disturbance of the ganglion cells in the substance of the cardiac tissues.

Those who believe in the myogenic theory of all cardiac disturbances will point to the fact that paroxysmal tachycardia is a not uncommon result of myocardial disease, inflammatory or degenerative.

In a case such as the above there was no evidence of any myocardial disease, past or present. We feel, therefore, that some explanation must be given of the fact that such a disturbance can arise in a heart presumably healthy as regards its tissues. It is difficult to imagine that one small area of the myocardium could have been infected by itself in such a way as to lead to serious cardiac failure, and that no other signs of an infection either of the system generally or of the heart could be detected. The lesion may have been a developmental one, i.e. a nest of cells intended for the sino-auricular node, the 'pacemaker of the heart', may have become stranded in some other part of the auricular wall and developed there its function of stimulus production. Such an explanation may account for the phenomena present, but it is at present without any proof.

The prognosis will depend first of all on whether the tachycardia persists or comes to an end. In some cases the tachycardia has persisted, and has been accompanied by signs and symptoms of cardiac failure leading to a fatal termination. This fact must be kept in mind when the question of prognosis is raised. On the other hand, the attacks may cease and the patient may be restored to sound health and the heart to a normal state. The question arises as to whether the heart completely recovers after frequent and prolonged attacks, and after signs of cardiac failure have been present. As regards the above case, the illness was too recent

for me to speak definitely of the ultimate prognosis. In the absence of recurrent attacks I should be inclined to consider the prognosis as decidedly hopeful. In the year 1895 I saw a very similar case of paroxysmal tachycardia, which was under the care of Dr. Herringham and has been published by him,[1] the patient being a girl of eleven years. In 1913 I learned that she was alive and engaged in active work, so that her heart has stood out satisfactorily for eighteen years after the illness. In considering the question of prognosis one must bear in mind the wonderfully recuperative powers of the youthful heart. In cases of paroxysmal tachycardia associated with organic disease of the heart, the rapid action is to be regarded as a part of the symptomatology and not as the cause of the cardiac disorder.

The question of treatment is a difficult one. Just as an attack may be excited by almost any form of disturbance, mental, mechanical, reflex, &c., so an attack may be checked in a similar manner. At most what is secured is a temporary cessation of the activity of the irritable area in the heart. Vomiting often seems to be effective, as in the above case, if brought about at the right time, probably when an attack is tending towards cessation. Bromides and other nervine sedatives may prove useful in quieting an excited patient and giving the heart a better chance to settle down. If one happens to be giving medicine at the time an attack ceases, the conclusion is often drawn that the special drug has cured the

[1] *Clin. Soc. Trans.*, vol. xxx, 1897.

tachycardia, but no drug will stand the test of experience in any such action. Rest, sedatives, attention to the various functions of the body, and the removal of any source of reflex disturbance will tend to induce the best conditions for a steadying down of the irregular action of the heart.

In the above case I tried the effect of digitalis in large doses and over a prolonged period. The doses given were larger than I have ever used for a patient of this age suffering from chronic carditis, and in fact I should hesitate to use such doses in the case of a heart organically diseased. Here we seemed to be dealing with a healthy myocardium. The object aimed at in this case by means of digitalis was to diminish conductivity and so check the passage of the rapid and abnormal auricular impulses through the heart. It was hoped by this means to steady the mechanism and so to give the left ventricle an opportunity of acting more slowly and contracting more vigorously. This object seemed to have been secured as shown by the rapid disappearance of the signs of cardiac failure at an early stage. But the effect was only temporary, and, although the heart recovered rapidly when given a period of rest, the tachycardia tended to recur as soon as the effects of digitalis had passed off. This raised the question as to whether it was possible to dull the excitability of the abnormal focus and check a local habit by giving larger doses of digitalis and over a longer period. This procedure was tried and was followed by a steady diminution in the frequency

and the duration of the paroxysmal attacks. So far the improvement has been continuous and progressive. I do not know whether this line of treatment can be successfully adopted in other cases of the same nature. There is this to be said in its favour, that it recognizes the cause of the disturbance in an abnormal centre of stimulus production and is directed to the removal of the effects of that disturbance through the known action of digitalis in other cases of disordered rhythm, e.g. auricular fibrillation. On the other hand, the cessation of the tachycardia may have been approaching from other reasons than the administration of digitalis. I am quite content to leave it at that for the present, but should certainly adopt the same treatment in any other persistent case of paroxysmal tachycardia.

SECTION C

ORGANIC HEART DISEASE

CHAPTER XI

INTRODUCTORY

After the age of four years rheumatic infection is common throughout the whole period of childhood and adolescence. During infancy, i.e. before the end of the second year, one rarely, if ever, meets with rheumatic infection or acquired rheumatic carditis. I have met with cases of rheumatic fever at the ages of four and five years which terminated fatally. In these, signs of cardiac involvement during life were well marked in the form of dilatation, valvulitis, and pericarditis, and cardiac changes were found post-mortem of a characteristic nature. While the presence of carditis undoubtedly contributed to the fatal issue, the fact that there was also a severe general toxæmia must be kept in mind. It is distinctly rare to find a first attack of rheumatic fever terminate fatally in childhood.

The frequency of rheumatic infection, as well as the multiplicity of the symptoms of that infection, are now emphasized by all authorities. Right on through childhood and youth the tendency to rheumatism is always present in certain predisposed

individuals, and the infection may be manifested by a definite attack of rheumatic fever, or by slight pains in the limbs, the so-called 'growing pains' of childhood, or tonsillitis, or a stiff neck, or slight arthritis, or other apparently trifling manifestation.

The importance of this infection in childhood lies in the fact that the heart is specially liable to be affected. Whether it be acute rheumatic fever, or subacute rheumatism, or rheumatism so slight that the symptoms do not attract notice, the risk of cardiac involvement is always present. While other rheumatic lesions tend to come and go, whether treated or untreated, and to leave no trace behind, the cardiac lesion tends to leave permanent changes. Other infections may occasionally lead to carditis, such as scarlet fever, pneumococcal infection, &c., but organic heart disease in childhood and youth is in the vast majority of cases due to rheumatic infection.

In many respects an acute attack of rheumatic fever is preferable to the more latent and unobtrusive forms, because in the former case treatment and care will probably be effective in saving the heart from infection or at least from permanent damage. It happens but too often that when the symptoms are slight treatment may not have been invited, or the tendency to heart trouble may have been overlooked. Some of the worst cases of cardiac disease in childhood which I have met with were those in which the rheumatic symptoms had been latent or so slight that no advice was sought until the patient

was suffering from a broken-down heart. It must be recognized that rheumatic infection may be present in such a latent form that the patient is never laid up for a day until the symptoms of heart failure have compelled attention. This fact emphasizes the importance of early and prolonged treatment of all rheumatic infections in early life, more especially as preventive and curative treatment give such satisfactory results.

In dealing with the subject of organic heart disease we may be taken as referring to the result of rheumatic infection unless otherwise stated. Similarly, when a case of acquired heart disease is met with in early life it may be assumed that it is due to a present or a past rheumatic infection, in the absence of definite evidence of other causation. Chorea and heart disease are often associated, and experience has taught us that both have a common origin in rheumatic infection. Yet they may be found separately or in combination in patients who give no history of having suffered from definite rheumatic fever or from obtrusive signs of rheumatic infection.

We view the importance of rheumatic infection according to the extent to which the heart has become affected. Fortunately in many cases an acute infection (rheumatic fever) may run its course without any involvement of the heart, and this result undoubtedly depends very largely on the care with which thorough treatment is carried out. When the heart has become involved the results vary

greatly in different cases. Clinically we meet with various types of cardiac disease. It is impossible to give a complete classification of all forms, but the following are some of these types:

1. Those in which a mild attack of valvulitis or myocarditis subsides without leaving any permanent damage.

This is the result always aimed at in our treatment and most frequently secured after a first attack. With each successive reinfection the chances of a complete cure become less and less. The signs of a slight lesion are unfortunately not always clear, but certainly in early life one can often observe the gradual disappearance of a systolic murmur which seemed to be due to a lesion of the mitral orifice, or of cardiac dilatation which appeared to be due to myocarditis, after a definite infection. The persistence of a murmur must not be taken as evidence that the heart has been functionally damaged. I find it difficult to state what the results of a slight pericarditis are, because this lesion occurs chiefly in connexion with the more grave forms of carditis and with recurrent attacks of rheumatic infection. So far as my own experience goes, there is reason to believe that an attack of slight pericarditis may be completely recovered from.

2. Those in which some permanent change in the valves or the myocardium or both is established, which does not produce any symptoms for years—ten, twenty, or more—but which is eventually followed by signs of cardiac failure.

A typical example of this class is seen in connexion with mitral stenosis, in which the orifice of the mitral valve becomes gradually narrowed. The characteristic presystolic murmur is not developed as a rule until later childhood or youth. Even after the murmur is well established, cardiac symptoms may be entirely absent for years. In the case of other lesions the result is often the same, so that it may be laid down as regards a large class of cardiac patients that while the rheumatic infection occurred during childhood, and the heart affection was established at the same time, the symptoms of cardiac disablement do not appear until much later in life. As a matter of clinical experience it will be found that such a case seldom goes on for years without recurrent rheumatic infection, which adds to the cardiac lesions.

3. Those in which the valves or muscular structures are so markedly affected that symptoms of cardiac distress or weakness are permanently established.

After a first, or even a second, attack of rheumatism this is by no means a frequent result. It is rarely seen after a purely valvular lesion, but is to be taken as an indication of a severe affection of the myocardium. In the case of a mitral lesion, the capacity of the myocardium to overcome any mechanical difficulty is usually such as to prevent the occurrence of signs of cardiac failure, and aortic regurgitation can be met in the same way. But an extensively diseased myocardium cannot hypertrophy to any

extent, and the more work it has to do the more marked and the more constant become the symptoms of cardiac distress.

4. Those in which a chronic rheumatic infection persists with subacute relapses, progressive carditis, and the development of symptoms of gradual heart failure.

This chronic or relapsing type of carditis is one of the most serious forms in childhood, and often one of the most intractable. It leads to continuous or interrupted changes in the valves, the pericardium, and the myocardium, at first inflammatory and exudative, and later on fibrotic. The other rheumatic manifestations may be so slight that, amongst the children of the poor at any rate, they are entirely overlooked. One can but assume that in these cases there is some focus of infection in the body, often hidden and undetectable by our present means of investigation, which pours out at intervals, or constantly, a supply of organisms or toxins leading to cardiac inflammation and degeneration.

5. Those in which the pericardium has become adherent to surrounding structures and the cardiac action is greatly impeded in consequence.

This is one of the most crippling forms of heart disease met with in early life. The heart's action is so impeded by the adhesions that the circulation is carried on with difficulty. Both signs and symptoms of heart failure are developed early. Some of the largest hearts met with in childhood are those which have become dilated and hypertrophied as

the result of adherent pericardium. If the heart is incapable of hypertrophy, as in cases complicated by extensive myocarditis, exhaustion of the cardiac muscle is not usually long delayed.

6. Cases in which either a specially virulent form of rheumatic infection, as Poynton thinks, or a superadded infection, as others think, has attacked the endocardium, leading to ulcerative endocarditis.

This is a rare condition in early life, but it is advisable to keep the possibility of its occurrence in mind because it is extremely fatal and it may not present striking objective or subjective symptoms. The patients are usually the subjects of pre-existing heart disease, with damaged valves, to which is added a fresh infection streptococcal or staphylococcal in nature. There may be extensive ulceration of one or more valves, with few physical signs, and in addition to the cardiac changes, a general blood infection is present, often accompanied by hæmorrhages, the result of thrombosis and embolism.

It has been the custom to consider the subject of cardiac disease in childhood chiefly from the valvular point of view. A murmur was heard and therefore heart disease was diagnosed. A case was labelled as one of ' mitral disease ' or ' aortic disease ' according to the valve which was affected, and treatment supposed to be appropriate to a mitral or an aortic lesion was ordered. Another large class of patients was labelled as suffering from ' irregular heart ' and the treatment supposed to be appropriate to that condition was ordered. This was largely the result

of the application of the methods employed in connexion with cardiac symptoms in adults to similar symptoms in childhood and youth. But if these methods were faulty and wrong in connexion with adult heart disease, they are doubly so in connexion with disease in early life.

In childhood we have to deal in the great majority of cases with the active and developing lesions of carditis, and in the minority with the results of these lesions in the form of an impaired functional power of the heart. In adult life, on the other hand, we have to deal in the great majority of cases with the effects of past carditis and chronic degenerative changes, and in the minority with the lesions of active carditis. The significance of the symptoms in early life is often entirely different from what it is in later years. While in connexion with adult cases it may be advisable ' to hope for the best and prepare for the worst ', this is not the spirit in which to approach cardiac disease in children.

The clinical division of cardiac inflammations into endocardial, pericardial, and myocardial is apt to be too much emphasized. It has a bearing on the differentiation of the various physical signs which may be present, but not on the prognosis or treatment. The physical signs accompanying these forms of inflammation are evidence of rheumatic infection, but as a rule it is the patient who requires treatment and not the heart directly. The endocardial lesion (valvulitis) may be very noisy in character, but may be quite trifling as regards its significance. Inflam-

mation of the pericardium is chiefly of importance as an indication that in all probability a severe and generalized carditis is present, and as a possible cause of external adhesions forming between the pericardium and the chest wall. The really important question as to endocarditis and pericarditis is as to their effect on the functional power of the myocardium. The myocardium is the most important and the most silent of the cardiac tissues in connexion with rheumatic infection. When we come to the fundamental factors in maintaining the functional efficiency of the heart, we find that the condition of the myocardium is the chief. When we look for the essential driving force in maintaining an efficient circulation we find it is the muscle of the left ventricle. As the result of disease other parts of the heart may have been affected and may have been functionally damaged, but the really important question is, what effect does this have on the driving power of the left ventricle? The splitting up of the various cardiac lesions into endocardial, pericardial, and myocardial is not of any real value in determining the course of the disease and may be misleading. What is essential is to find out how far those various factors are present and how far they are affecting the action of the left ventricle.

The myocardium in childhood, as in later years, has the functions of contractility, excitability, rhythmicity, tonicity, and conductivity. Both in acute affections, and as the immediate result of them, these functions are not usually disturbed in a manner

which attracts special attention, or which differs from the conditions found in any other acute illness. Judging from the very different conditions found in adult life, it would appear that degenerative or fibrotic changes in the heart muscle cause much more disturbance of these functions than acute inflammation. The myocardium may have been acutely inflamed and yet may be altogether silent so far as any evidences of its functional impairment can be detected. This is one of the problems of heart disease in childhood, to decide whether the myocardium has suffered and whether in the future there lies the possibility of impaired ventricular action.

CHAPTER XII

GENERAL SYMPTOMATOLOGY

Significance, Origin, Character, and Persistence of Symptoms

In considering the functional disorders and disturbances of the heart, we saw that they took the form of variations in rate, irregularities in rhythm, murmurs, dilatation, &c. In dealing with the symptomatology of organic heart disease we shall find that similar signs are present. For instance, an extra-systole may be present at times in a heart otherwise normal in every way. An extra-systole may also occur in a heart manifestly diseased as

the result of rheumatic infection. Naturally more importance is to be laid on this sign in the latter case. To use a simile of Wenckebach's, there is a great difference between a man who now and then makes a false step, stumbles, or limps because he has some disease of the lower extremities, and another who, although his legs are quite sound, stumbles when he is walking over rough ground, sharp stones, or when he steps into a deep hole. The question of the **significance of the signs** and symptoms of organic heart disease is of the greatest importance but also of the greatest difficulty. In the case of functional disorders we have taken the view that, as a rule, the significance of the signs and symptoms, so far as the heart is concerned, is not great. In connexion with organic heart disease we find the symptomatology is so varied that its significance can only be estimated in individual cases and after a comprehensive consideration of all the personal conditions. One point has been strongly emphasized by Mackenzie, viz. that the significance of one sign or symptom should not be exaggerated. In the past it has been too much the custom to examine the heart for any evidence of disease, and on the discovery of even one sign, to say that the patient has a weak heart. More especially did this apply in the case of the discovery of a cardiac murmur. Sometimes care was not taken even to decide whether the murmur was congenital, or functional, or acquired and organic. The real significance of the sign was not examined into but it was inter-

preted as evidence of a weak heart, and in many cases measures have been taken which would at least have the effect of preventing the patient from ever having a strong heart in the future. The significance of the symptomatology of organic heart disease is at present by no means thoroughly understood. Our knowledge of the subject is being constantly added to. But we have at least reached this stage, that an ominous significance is not to be attached to a murmur or an irregularity simply because the observer does not understand what it means.

As regards the **origin of the physical signs** of heart disease, we can ascribe them in the vast majority of cases to rheumatic infection of the heart. Organic heart disease begins with the development in the fibrous or muscular structures of an inflammatory process, the result of the rheumatic poison directly attacking the heart. This condition, which is obscure at first, is gradually revealed by the appearance of certain signs and symptoms which will be described more fully later on. This origin of the signs of organic heart is in marked contrast to that of functional disturbances, which, as we have seen, lies for the most part in factors entirely outside the heart.

The **character of the physical signs** of heart disease in childhood is in many respects not unlike that of functional disturbance. We meet with alterations in rate, chiefly in the way of an increase. We meet with murmurs which are definitely of two kinds, the valvular and the pericardial, but murmurs the

result of dilatation and produced at the valves may be associated with cardiac disease as well as with cardiac disturbance. Organic valvular murmurs are indications of structural changes in the valves themselves. In the case of certain organic murmurs, e.g. the mitral presystolic and the aortic diastolic, we have characteristic sounds which are both in time and in character unlike anything heard in connexion with functional disturbances. In the case of other organic murmurs, usually systolic in time, there is often a characteristic roughening or a musical element which is not present in the case of functional murmurs. Still the fact remains that many systolic murmurs are found in children which present no criteria by means of which one can say that there is organic valvular change. So we find generally that, taken singly and without associated cardiac changes and without associated general symptoms, the signs of organic heart disease are not at all unlike those of functional disturbance.

There is, however, a marked contrast between the two in the matter of **persistence and stability.** The functional signs are subject to great variation from day to day, with change of position, and with the state of the general health. The organic signs, on the other hand, are much more persistent under the above conditions. Viewed from another standpoint the signs of organic disease are much less persistent in early years than in adult life. There may be slow improvement with a gradual diminution of all signs, or possibly even disappearance. Some

of them, e.g. murmurs, may persist for years and then fade away. In the case of progressive disease the tendency is for the signs gradually to increase and alter as the result of fresh cardiac lesions.

CHAPTER XIII

IRREGULARITY OR ARRHYTHMIA

(A) Sinus Irregularities, (B) Extra-systoles, (C) Paroxysmal Tachycardia, (D) Auricular Fibrillation, (E) Auricular Flutter

In the past it was not uncommon when a heart was found to be irregular in its action to start a course of treatment for the cure of this irregularity. The irregularity was looked on as the disease although its origin and character were quite unrecognized. Modern progress has changed all that. We now seek first of all to determine the nature of the irregularity, and thanks to instrumental records this can be done with a great measure of exactness. The next thing is to determine the site of origin of the irregularity, whether it is outside of the heart or, if in the heart, in what special part. This also can be accomplished in the great majority of cases. The next point to determine is the significance of the irregularity, and on this subject knowledge grows daily. The last question is the treatment of the

irregularity, and speaking generally it is only in few cases in childhood that the irregularity has any bearing on the treatment of the patient.

In connexion with functional disturbances we have already spoken of cardiac irregularities. In my experience they are more commonly met with as a part of a functional disturbance than in connexion with organic disease. Certainly they do not bear in childhood the same outstanding position in symptomatology that they do in adult life and more especially in old age. There are certain reasons for this. One is that many forms of irregularity are diminished or abolished when the heart rate is increased. In childhood, acute and progressive cardiac disease is accompanied as a rule by a much more rapid action of the heart than occurs in later years. Again, many cardiac irregularities are associated with advanced changes in the heart muscle, and with degenerations, which have not had time to develop as a rule until after adult life has been reached.

Clinically it is to be noted that during the acute stages of rheumatism and the active stages of cardiac disease irregularity of action is less likely to appear than in the non-febrile and quiescent periods. In the acute stage the increased rate is sufficient to remove certain forms of irregularity, e.g. the extrasystole. A slowly beating heart is a much more likely source of irregularity than a rapidly beating one.

Irregularities can now be classified, and I shall

consider the chief varieties that are met with in early life:

(*a*) Sinus irregularities.
(*b*) Extra-systoles.
(*c*) Paroxysmal tachycardia.
(*d*) Auricular fibrillation.
(*e*) Auricular flutter.

(*a*) **Sinus irregularities** are those which arise in the sino-auricular node, the tissue which acts as the 'pacemaker of the heart' in Lewis's phraseology. Another term is vagal irregularities, because the vagus nerve controls this tissue, and the commonest forms of irregularity produced here are due to vagal disturbance. Consequently a sinus irregularity is not to be regarded as an evidence of heart disease, and the majority of such disturbances fall into the category of functional disorders which have been considered in a previous section. So common are they in healthy young people with healthy hearts in the form of an increasing rate with inspiration, and a decreasing rate with expiration, that Mackenzie has raised the question as to whether they may not be a test of recovery after an attack of carditis. He has come to look upon the presence of this irregularity, when the cardiac rate has fallen below 70 beats per minute, as an evidence that the heart has escaped infection. He has never seen this irregularity present in an acute infection or with a progressive lesion of the heart muscle. He merely suggests that further tests should be made as to the

value of this symptom from the point of view stated above. I have tried to test this suggestion, but have been unable either to confirm or refute it. *Prima facie* I am doubtful as to its value, because just as Mackenzie hesitates to diagnose heart disease on one symptom only, so I should hesitate to assume that the heart has escaped damage on one symptom only. The essential accompaniment of this symptom is a slow pulse and that of itself is one of the most definite signs of cardiac recovery in early life, just as the rapid pulse, with which any sinus irregularity disappears, is one of the most definite signs of active and progressive carditis.

One thing must be kept in mind, namely, that this form of sinus irregularity in which variations occur in the length of diastole, the diastolic variation affecting both auricles and both ventricles, is not a sign of organic heart disease. It is a phenomenon associated with cardiac action in childhood and youth, which in the majority of cases is so slightly marked as to escape observation, but in certain cases will be so pronounced as to attract the notice of the clinician as a definite irregularity of the pulse which can be felt.

There is another variety of sinus irregularity which Dr. Lewis describes as a 'phasic variation of pulse rate, in which a retardation and subsequent gradual acceleration of the whole heart occurs'. He says that the change may be spread over ten, fifteen, or more seconds, and may be repeated regularly or only from time to time. As a clinical condition this

is not often met with, and even if present it would not be so pronounced as to arouse any question of irregularity in the examiner's mind. I have met with one marked case due to what is, according to Dr. Lewis, the most common cause, viz. the administration of large doses of digitalis. It was in the case of a boy aged ten years, suffering from paroxysmal tachycardia, to whom large doses of digitalis were given for a prolonged period. When the heart did slow down there was a diphasic variation in the pulse rate. At one time it would be beating at 45 per minute and then would change to 75 (Fig. 43). As the change would occur every six or eight beats, the sense of irregularity was very marked when one was feeling the pulse. Apart from this cause, full dosage with digitalis, I have not met with this condition, and with the removal of the cause in the above case the irregularity soon passed off.

The next group of cardiac irregularities is associated with the development in the heart of a new starting-place or new starting-places for the cardiac contraction, so that the normal contraction at the sino-auricular node is either supplemented or replaced entirely by the new rhythm. The extra-systole is the commonest type. Another illustration is seen in paroxysmal tachycardia, and a third in auricular fibrillation. Auricular flutter is a fourth variety.

(*b*) **Extra-systoles.** As regards their frequency in cases of organic heart disease they may be said to be rare during the first decade and more common

during the second. Even during the first ten years of life they are, in my experience, more frequently associated with functional disturbance than with organic disease. During childhood one of the chief features is the fugitive character of extra-systoles associated with cardiac weakness or cardiac disease. Thus they are not uncommon occurrences during the convalescent stage of pneumonia when the pulse rate is slowing. During the active carditis of rheumatic infection they may be present at intervals and disappear with the subsidence of the acute inflammation. In all these cases they come and go in the most surprising manner, not only from day to day, but from hour to hour. As already stated, an increase in the pulse rate from any cause, such as excitement, will usually lead to their disappearance.

The importance of the recognition of extra-systoles is not from their bearing on the patient's condition, which is negligible, but in preventing the physician from regarding the irregularity as a serious one. The common 'missed beat' or 'intermittence' felt in the pulse is usually due to an extra-systole. It is definitely recognized on auscultation of the heart by the presence of one premature sound or two premature sounds, according as the aortic valves have not or have been opened by the premature contraction. It gives a characteristic tracing with the polygraph (see pp. 31 et seq.). As already stated, it is not so common in organic disease as in functional disturbance, and it is much less persistent in the

former than in the latter condition. The explanation of the occurrence of extra-systoles would appear to be that the function of excitability in the cardiac muscle, present in all parts, is increased in the subjects presenting extra-systoles. This abnormal excitability, as long as it is limited to the production of extra-systoles in a heart acting at or below the normal rate, is not of any importance from the prognostic point of view whether it occurs in a diseased heart or as the result merely of functional disturbance. I cannot say that the presence of extra-systoles has aided me in the diagnosis, prognosis, or treatment of heart disease. They do not seem to attract the attention of the young patient in any way, nor do they produce any symptoms. Until some more definite knowledge has been obtained as to what, if any, significance should be attached to extra-systoles, I am inclined merely to take note of their presence.

(*c*) Of more importance is the increased excitability of the heart as manifested in the condition of **paroxysmal tachycardia.** No irregularity of the heart is manifested during the paroxysm, but only at the onset and after the termination. This condition has been fully discussed elsewhere (p. 78) and is distinctly rare during the first two decades of life. It may occur in association with other signs of organic heart disease, and seriously affect the functional power of the heart, as already described.

(*d*) **Auricular fibrillation.** This is a special form of increased excitability of the auricular walls which

leads to marked irregularity of the pulse. The importance of the subject in connexion with adult life is now fully recognized. Its importance in early life is equally great but is diminished by the fact that examples of the condition are but rarely met with. Dr. Lewis has met with but four cases under the age of twenty years out of a total of 116 cases. The conditions of its occurrence, namely, an altered nutrition of the auricular muscle (Mackenzie), are probably the same at all ages. Clinically the gross irregularity of the pulse, in which the beats vary constantly in force and spacing, is a striking feature, while other signs of cardiac disease are always present. There is nothing like this met with in the irregularities associated with functional disturbance.

In a case recorded by Carey Coombs and myself we found the irregular cardiac action of auricular fibrillation and a ventricular venous pulse in a child of five years shortly before death, which was due to rheumatic fever and acute carditis. The duration of the whole illness was six days. The explanation of the auricular fibrillation in this case seemed to lie in the condition of the left auricle, which was very extensively diseased as the result of acute inflammation and fatty change. Mackenzie detected the condition in a young man recovering from a mild attack of rheumatic fever. The attack passed off after some hours and complete recovery followed with no evident lesion of the heart. Price has found auricular fibrillation in a fatal case of diphtheria. The onset of auricular fibrillation in connexion with

acute disease is rare, and it commonly occurs in a heart which has been for a long time extensively diseased or degenerated. Hence the infrequency with which it is met with in early life. The youngest case I have seen was a girl of thirteen years, and she developed the condition after suffering for many years from rheumatic carditis. The patient was under the care of my colleague, Dr. Leonard Guthrie, to whom I am indebted for permission to refer to it.

Case XI. Patient had had her first attack of rheumatic fever when she was six years old and was in hospital for four months. Other attacks had followed when she was 7 years, 7½ years, and 10 years old, and had been severe. The heart had been involved and she had become quite crippled as regards making any but the most moderate amount of exertion. At the age of eleven she had had an attack of acute cardiac failure, with great dyspnœa, pulmonary œdema, and pericarditis. At that time it had been noted that the cardiac action was very rapid and very irregular, but the nature of the irregularity was not determined.

The present attack had begun two weeks previously with increasing dyspnœa and vomiting, and the legs had become swollen and painful. On admission she presented the typical appearance of acute cardiac failure, viz. dyspnœa and orthopnœa, cyanosis, restlessness, and marked œdema of the lower extremities. The heart was much enlarged both to right and left, and the heaving præcordium suggested the condition of adherent pericardium. There

was a loud systolic murmur, practically replacing the first sound, and loudest over the left side of the heart and in the axilla. Owing to the rapid action it was impossible to determine accurately any other murmurs, but one occurring during diastole seemed to be audible occasionally.

The cardiac action was rapid and irregular. The irregularity was of a very marked kind and strongly suggested that associated with auricular fibrillation. A tracing of the radial artery showed the characteristics of this condition, a total irregularity as regards time and force of beats. A venous tracing showed complete absence of the auricular wave, while *v* and *c* were well marked. As estimated by the tracings, the number of cardiac pulsations reaching the wrist was from 125 to 130 per minute. Auscultation of the heart showed a more rapid rate than this.

Leeches were applied, stimulation with brandy was given, and the patient was ordered five drops of tincture of digitalis and four grains of theocin sodium acetate every four hours. Diuresis set in quickly and the amount of urine passed in the following three days was 36, 41, and 51 ounces respectively, after which the amount became normal. This speedy action must, I think, be ascribed more to the theocin sodium acetate than to the digitalis, but it led to a rapid diminution in the œdema.

The general symptoms were improving before the cardiac rate was noticeably affected, but on the sixth day of treatment it had fallen to 102, and then on consecutive days to 100, 90, 84, 84, 66, 60, 60,

58, 58, when the digitalis was stopped. The cardiac action was still irregular but very much less so than before, and the force of the beats as judged by the pulse had greatly increased. The child was quite comfortable and could lie down without any dyspnœa. During the fifteen days she had taken 4½ drams of tincture of digitalis.

There was no return of the acute symptoms of cardiac failure, and the cardiac irregularity and

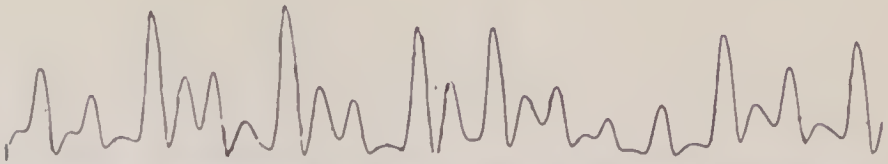


Fig. 44. Auricular fibrillation. Girl of 13 years. Rheumatic carditis. Radial tracing. Pulse rate = 125–30. The grossly irregular type of pulse characteristic of fibrillation is present.

rapidity were improved for some weeks. Another course of digitalis was begun at the end of six weeks owing to increased cardiac disturbance and was continued for three weeks, 5 minims of the tincture being given thrice daily. This was found to be the dose which would best maintain the efficient action of the heart for the needs of the patient when leading a quiet life, and beyond this the cardiac power could not go. This dose also served to steady the cardiac action and to maintain a rate of from 70 to 80 beats per minute.

The first tracing (Fig. 44) shows the markedly irregular pulse some days after admission, which cannot be associated with any form of cardiac irregularity save that of auricular fibrillation. A venous tracing (Fig. 45) taken at the same time confirms this, as it is ventricular in type, with no trace of auricular contraction. Nine days later the pulse rate had fallen and the tracing (Fig. 46) shows

Fig. 45. Auricular fibrillation. Same case, same date. The upper tracing (venous) is of the ventricular type, i.e. there is no trace of an auricular wave.

a much more regular action. Extra-systoles had now become the leading feature (Fig. 47). Eleven days later the pulse was very satisfactory from the point of view of rate, strength, and regularity (Fig. 48).

A careful study of this case showed that the signs and symptoms of auricular fibrillation as observed in adult life were exactly reproduced, and further, that the effects of digitalis were the same. The irregularity was here a very important symptom,

FIG. 46. Auricular fibrillation. Same case, nine days later, after digitalis. Pulse rate = 55–65.

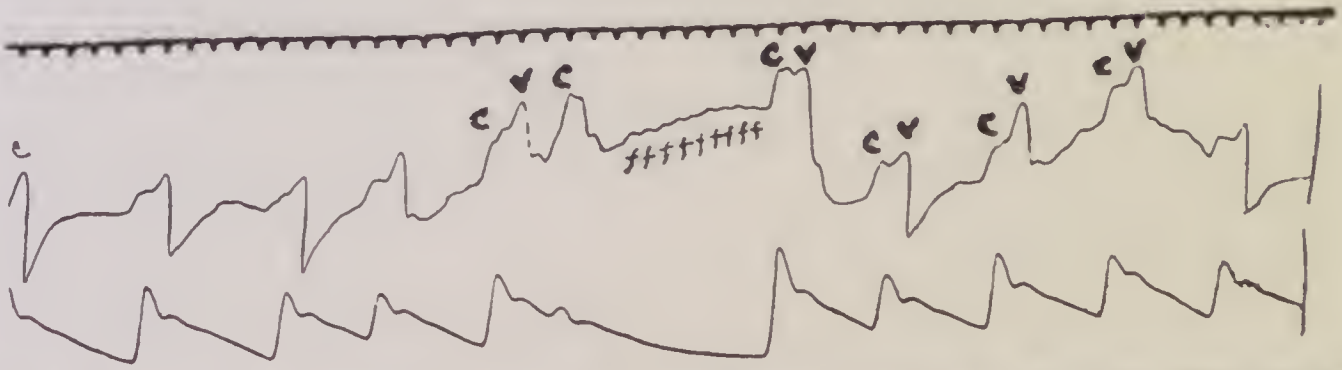


FIG. 47. Auricular fibrillation. Venous and radial tracing. Same case, same date. Extra-systoles seen in radial pulse. The ventricular type of venous pulse is still present and the auricular fibrillation is indicated on the tracing (*ff*).

Fig. 48. Auricular fibrillation. Venous and radial tracing. Same case, eleven days later. Pulse is now much more regular. Occasional extra-systole. Sinus irregularity.

indicating the cause of the cardiac breakdown and thus the line of treatment.

(*e*) **Auricular flutter.** This condition was so named by MacWilliam, who found it in the course of his physiological experiments, and its clinical recognition is due to Ritchie, who demonstrated it clearly by means of the electro-cardiograph. So far only four cases have been published between the ages of ten and nineteen years (Cowan). Auricular flutter is characterized by an increased rate of cardiac action in which the auricles contract at a much greater rate than the ventricles, but usually in some definite ratio such as two to one, four to one, &c. The ventricular action and the pulse may be perfectly regular, and of this an example is recorded later on in connexion with acute general carditis. On the other hand, the pulse may be markedly irregular and very similar to that in auricular fibrillation, with which condition in fact flutter has been confused until quite recently. It is only in exceptional cases that the condition can be clearly made out by means of the polygraph, and the electro-cardiograph is usually necessary for a clear demonstration of auricular flutter.

CHAPTER XIV

RAPID ACTION OF THE HEART

In acute rheumatic infection the heart rate is increased, but in many cases the increase is not out of proportion to the fever and the toxæmia present. An average rate may be put down as from 110 to 115 in a child of ten years. When the rheumatic attack is specially severe, or when there is active carditis, the rate may be a good deal higher, 125 to 160. But we cannot tell by the pulse rate alone that active carditis is present, and in considering the pulse rate as a symptom attention must be directed to other manifestations of disease and disturbance. We have to take into consideration any other signs which point to a severe infection and look out for other signs of cardiac involvement. We have also to reckon with other causes of cardiac disturbance, such as a nervous and excitable nature, pain, sleeplessness, and fever. These factors may markedly affect the heart rate, and it is only when they have been excluded that one can settle on the heart itself as the cause of the disturbance. When the carditis is responsible for the tachycardia we shall expect to find the latter persistent under changes of position, variations in temperature, and sleeping and waking.

In cases of chronic carditis I believe that very great importance is to be attached to the presence

of a rapid action of the heart. One will often find that a definite rate with regular rhythm is established and persists. An average rate is from 115 to 125 beats per minute. This rhythm once established may go on for weeks or months, with very slight variations under the ordinary tests, sleeping or waking, sitting up or lying down, slight pyrexia, &c. Such a condition is not found as the only manifestation of rheumatism or carditis; if it were the diagnosis would be open to question. It is usually associated with other signs of cardiac disease, e.g. dilatation, valvular murmurs, &c. It is often associated with the presence of rheumatic nodules in various parts of the trunk and extremities. It occurs in cases of chronic or relapsing rheumatic infection, as shown by slight pyrexial attacks, fleeting pains in the joints, or fibrous tissues, erythematous rashes, &c. It often occurs in connexion with adherent pericardium where progressive cardiac change is going on. From these associations I have been led to regard a persistently rapid heart rate in chronic cardiac disease as a sign of great importance. Its presence in some cases is probably due to active carditis and persistent rheumatic infection, in others to progressive myocardial changes increasing the excitability of the heart. The significance of this persistent tachycardia from a prognostic point of view is serious. Not that the prognosis has to be based on this sign alone, for other evidences of cardiac disease are always associated and will have to be taken into consideration. In some cases we

find this persistently rapid pulse with a considerable degree of pyrexia, the temperature range being of the hectic type, and with manifest prostration. These conditions, when associated with the signs of definite heart disease, suggest a condition of septicæmia which may or may not be accompanied by ulcerative endocarditis.

CHAPTER XV

SLOW ACTION OF THE HEART

After an attack of rheumatic fever or of acute carditis one of the most satisfactory symptoms is a fall of the cardiac rate to normal. We regard this as indication that the condition of the heart is improving or that the muscle has not been seriously affected. A reactionary slowing of the pulse, after a period of marked rapidity, may go even further, so that the rate is reduced to 50 or 60. This taken by itself is not a sign of bad significance, as the condition will probably be temporary.

The occurrence of extra-systoles which are not sufficiently strong to affect the pulse may suggest an abnormally slow action of the heart. This source of error will be avoided if the heart is always examined as well as the pulse. On auscultation the sound or sounds accompanying the extra-systole will reveal the true cardiac rate.

The more special form of slow pulse which is associated with heart-block is not common in early life. As the question is one of the conduction of impulses, it may be assumed that the junctional tissues (auriculo-ventricular node and bundle of His) are not functionally disturbed by rheumatic infection in ordinary cases. It is difficult to see how they can fail to be anatomically affected by the widespread inflammatory changes which are so often present. A few isolated cases of heart-block in early life have been recorded in association with lesions of the junctional tissues, e.g. tumours, diphtheritic inflammation, &c. Fleming and Kennedy have recorded a case of diphtheria in a child of ten years with complete heart-block. There was a ventricular rate of 46 to 54 and an auricular rate of 100 to 110. Post-mortem examination showed well-marked evidence of myocarditis, the *a–v* node and the first part of the *a–v* bundle being involved in the inflammatory process. This abnormal type of bradycardia, whether in acute disease or in chronic cardiac lesions, is not common in early life.

True bradycardia, i.e. the form in which all the chambers of the heart take place, a sinus bradycardia, is not common in early life in association with organic disease of the heart. Personally I have no records of its occurrence in a form sufficiently marked to call for notice.

CHAPTER XVI

DILATATION AND HYPERTROPHY OF THE HEART

Dilatation of the heart occurs with a frequency and a facility in early life which is not equalled in later years. I have watched a case of paroxysmal tachycardia during the slow period, when suddenly a paroxysm came on and the rate rose from 42 to 160 beats per minute. At the same time the heart dilated to the extent of over an inch as judged by the position of the apex beat. There were no symptoms of cardiac distress under either rate, unless the tachycardia was long continued. One could not help regarding the dilatation in this case as due to a self-regulating mechanism of the heart to cope with the disturbance of the cardiac action from the onset of tachycardia. In this case there was no organic heart disease discoverable, but a definite disturbance of the normal rhythm due to the presence of an ectopic centre of impulse formation in the auricle. If in such a case dilatation can be viewed as a compensatory mechanism, the same will probably hold good in cases of cardiac embarrassment from organic disease (myocarditis, valvulitis, pericarditis).

We have seen in considering the functional disturbances of the heart that dilatation is quite common in the case of children whose hearts are

healthy. The dilatation in such cases was ascribed to an atonic condition of the heart muscle, often of nervous origin. Reference has already been made in connexion with cardiac disturbances to an **apparent dilatation of the heart** which is really a **displacement.** In connexion with acute illnesses one may easily make this mistake, as in the following case.

Case XII. A boy of six years was sent into hospital with a diagnosis of acute rheumatism. He had been ill for three weeks, suffering from pyrexia, pains in the limbs and head, and diarrhœa. The boy looked extremely ill and showed signs of great prostration. He was markedly jaundiced, the liver was enlarged, and the motions were loose and offensive. The apex of the heart was felt beating in the 4th space one inch external to the nipple line on the left side. There was plural friction at the base of the left lung posteriorly. The diagnosis was by no means clear, but on the fourth day he was put on salicylate of soda, grs. x every four hours. Within twenty-four hours the temperature fell from 103° to 100°, and twenty-four hours later it was normal. There was no further fever.

The boy improved steadily, but the case did not quite correspond with the ordinary course of an attack of rheumatic fever with dilatation of the heart. In fact the dilatation of the heart stood out by itself as the one and only indication of cardiac involvement, and persisted unchanged after all other symptoms had subsided. On further examination it was found that the right border of the heart was

to the left of the sternum, as estimated by percussion, and that the note over the left lung was distinctly impaired while the breath sounds were weak, especially at the base. There is little doubt that the boy had suffered at some previous time from pleurisy or pneumonia leading to pulmonary fibrosis and displacement of the heart to the left. When he left the hospital a month later the heart was exactly in the same position as when he entered it. The acute illness was probably of an infective character, but there was no definite evidence of a rheumatic infection.

One must bear in mind the possibility of cardiac displacement, and of pericardial effusion, before deciding that an extension of the cardiac dullness or pulsation is due to dilatation.

The determination of the **extent of the cardiac dilatation** may be a matter of some difficulty. So at least it has appeared to me when several doctors have been asked to examine the same heart and have given very various estimates of the extent of the cardiac boundaries. In most children palpation will serve to indicate the apex beat, which is probably the best standard by which to judge the **size of the left ventricle.** If the left ventricle is dilated the apex beat is usually displaced upwards and outwards, not downwards and outwards as in later life. Percussion may be employed as an additional means of determining the left border of the heart, but this requires a considerable amount of practice. It is from this method that so many diverse estimates

of the amount of dilatation come, so that on this test alone I do not trust my own results, and far less those of others. It is by the method of percussion that those very extensive forms of dilatation are discovered which I must admit my ignorance of. If they really existed we should also be able to determine by palpation that the apex beat had also extended very far outwards, and this in my experience is rare. So that in deciding as to the amount of dilatation in childhood both palpation and percussion should be in accord, and if they are not the indications supplied by palpation are probably the more reliable.

The estimation of **right-sided dilatation** is also to be made by palpation and percussion. When the right side is enlarged the pulsation felt at the ensiform will probably be amplified and intensified. This is really the only area in which the right side of the heart can be palpated. An extension of the cardiac dullness to the right of the sternum may be made out, but in my experience it is not always a reliable test. The parts of the heart which are here accessible to percussion are the anterior surfaces of the right auricle, and the right ventricle, of which the former is probably the more important. When the right heart enlarges it may encroach on and displace the anterior part of the right lung in such a way as to come close to the chest wall. In this case the superficial cardiac dullness extends beyond the right side of the sternum and is easily defined on percussion. On the other hand, the dilated heart

may extend at a deeper level in the chest, the anterior surface of the lung may occupy its normal position, and instead of a superficial there is a deep cardiac dullness to be defined. This is by no means an easy task. Clinically, therefore, we find that while an extension of the superficial cardiac dullness to the right may be a valuable sign, on the other hand its absence must not be taken as excluding dilatation of the right heart.

In **acute rheumatic infection** dilatation is often present, but I think its importance and significance have been overestimated by many writers. Considering the frequency with which the hearts of healthy children will dilate, and the hearts more especially of nervous children, and the hearts of children suffering from various forms of acute pyrexial disease, I fail to see why dilatation in rheumatic carditis should be emphasized so strongly. In my experience it does not, as a rule, exceed in extent the dilatation met with in the above-mentioned conditions. It is not accompanied by any symptoms of cardiac distress, and varies considerably from time to time during the course of the acute illness. It is probably in the majority of cases due to the self-regulating mechanism of the heart adjusting its size to the needs of the circulation. Hence, taken by itself, dilatation is not to be regarded as necessarily a sign of evil omen.

A more serious form of dilatation is met with in connexion with an attack of **acute carditis**, in a previously damaged heart. This is seen typically

in association with pericarditis, a form of inflammation which is of importance not so much in itself as in being an indication of serious myocarditis. A previously damaged heart under the influence of a fresh rheumatic infection will often show marked dilatation, sometimes of the left side, sometimes of the right side, but more commonly of all the chambers of the heart. Along with this dilatation other signs of extensive carditis, and symptoms of cardiac embarrassment (pallor, restlessness, dyspnœa) and signs of cardiac failure (enlargement of the liver, pulmonary engorgement, œdema, &c.) will certainly be found. The strain on the damaged heart in such cases is manifested not only by the dilatation but by the thumping action of the left or right ventricle present, an overaction of the muscle which is an additional evidence of the embarrassed circulation.

In **chronic heart affections** dilatation may be present either alone or in combination with hypertrophy. The dilatation, taken by itself, probably indicates a mechanical difficulty in the circulation which requires to be met by a slight alteration in the size of the heart, usually of the left ventricle. For instance, in the case of aortic regurgitation a larger quantity of blood has to be thrown into the aorta and so the ventricle dilates. The extent of the dilatation comes to be a measure of the difficulty to be overcome. In many cases in childhood, when a certain degree of dilatation has once been reached there seems to be little change for

some years, in the absence of fresh attacks of carditis. Here it may be assumed that the difficulty has been successfully overcome. Dilatation of the left ventricle is therefore common in cases of myocardial weakness from disease, and in cases of circulatory difficulties the result of valvulitis (mitral disease, aortic disease).

While there may be a compensatory mechanism in connexion with dilatation, this sign is not to be regarded as one of strength but of weakness. In all cases of **heart failure,** whether associated with acute or chronic disease, evidence of cardiac dilatation will be present. Mackenzie regards dilatation as being due to a depression of the function of tonicity in the cardiac muscle, and probably this is the best explanation which has yet been given of the condition. Dilatation in this sense is an indication of one form of cardiac weakness. It has appeared to me that this function of tonicity is not established so firmly in youth as in later life, so that dilatation occurs more readily both in conditions of health and of disease. Further, this dilatation is not only excited more easily but it subsides more rapidly under conditions of health and disease. But if it is accompanied by signs of progressive carditis, by other signs and symptoms of cardiac failure, then dilatation has the same grave significance in early life which it has in later years.

Dilatation of itself may not suffice to overcome difficulties. Another property inherent in the heart

muscle is that of **hypertrophy**, and it is probably greater in the growing heart of a child than in the adult organ. Hypertrophy comes into play whenever there are circulatory difficulties to be overcome, and in childhood the chief difficulties arise in connexion with valvular disease and with adherent pericardium. Hypertrophy is not only an indication that there has been a call for further driving power on the part of the heart; it is also an indication that the myocardium has not been extensively damaged, for degenerated muscular tissue cannot hypertrophy. However extensively the other special cardiac tissues may have been affected, we may look on hypertrophy as a sign that the muscular tissues are healthy in whole or in part. At the same time it must be kept in mind that, as Mackenzie says, 'a hypertrophied heart is always an impaired heart, and however complete the compensatory hypertrophy may be, there will always be found a limitation in the field of response'. The process of hypertrophy is always a slow one. We do not find it occurring during acute rheumatic infections, or in cases of active carditis, but in long-standing cases of chronic heart disease with some mechanical difficulty in the circulation.

The most striking form of hypertrophy met with in childhood is in association with **adherent pericardium.** Whenever a very large heart is met with, the apex lying in the axilla, the right border extending well to the right of the sternum, the whole præcordial region pulsating and thrust forward, it

is usually safe to assume that pericardial adhesions are present and have led to the hypertrophy. Very striking hypertrophy may also be found as the result of **aortic disease,** usually regurgitation, and this will be found to affect the left ventricle chiefly. In cases of mitral regurgitation hypertrophy is as a rule much less marked, and this may be due to the absence of any necessity for hypertrophy or to the presence of myocardial changes in the left ventricle preventing hypertrophy. Hypertrophy of the right ventricle is seen chiefly in connexion with pulmonary stenosis (congenital) and mitral obstruction (acquired).

The **diagnosis of hypertrophy** is not to be made so much from the size of the heart as from the nature of the cardiac impulse. There is a characteristic thrust or heave against the palpating hand which is quite different from the ordinary impulse, whether that is weak or strong. The impulse is always much more diffused than usual, and often covers the whole of the greatly extended præcordial region. As Mackenzie points out, the thrust forward of the chest wall during systole is associated with hypertrophy of the left ventricle, while that during diastole is associated with hypertrophy of the right ventricle. The difference is due to the fact that the left ventricle pushes forwards in contracting, towards the apex more especially, while the right ventricle contracts away from the chest wall and rebounds in diastole. In cases of adherent pericardium I have not usually been able to make out this distinction.

The pulse in adherent pericardium is often misleading, for it may be small, ill sustained, dicrotic, or collapsing. The strong cardiac action seems to have a very poor result. This result, however, is often due to the fact that the left ventricle is so tied down that it is unable to empty itself and may even carry on the circulation with difficulty. The high-tension pulse, with hypertrophy of the heart, which is so common in later years, is seldom met with during early life, and then it is probably connected with that rare disease, granular kidney and arterio-sclerosis.

CHAPTER XVII

VALVULAR MURMURS

It is not going too far to say that by many the diagnosis of heart disease has often been made simply on the discovery of a cardiac murmur. It may have been functional or organic, or congenital, or extra-cardiac, but on the evidence of the stethoscope alone the child was pronounced to have 'a weak heart', or 'heart disease'. This fact has been brought home to me very frequently in connexion with the medical examination of school-children. In that branch of practice where patients are many and time is short, it is not uncommon to find that the advice has been given to parents to seek medical treatment for heart trouble in a child.

On examining these patients I find that the most common objective sign is a murmur audible in the præcordial region, and often nothing else suggestive of disease. These young people, simply because a cardiac murmur has been discovered, have been sent to the schools for invalid children and have been duly labelled as suffering from heart disease or a weak heart. The same thing often happens in private practice, where the discovery of a cardiac murmur is followed by extreme precautions lest something further should develop. These results are due to the faulty or incomplete teaching of the past that a cardiac murmur is definite evidence of heart disease, and necessarily entails cardiac treatment. Conclusions such as these are difficult to eradicate, although they are not based on experience or careful clinical observations.

The valvular murmur of organic disease is important chiefly from the diagnostic point of view. It is an indication that valvulitis is or has been present. It is evidence of past or present rheumatic infection of the endocardium. It must necessarily be considered along with the other signs of rheumatism and of carditis. It is not usually of any prognostic value. It does not directly affect the treatment of the patient, and far less is it to be regarded as requiring any treatment itself.

Valvular murmurs in early life are similar in cause and character to those met with in later years. They are the result of changes taking place in the valves during the course of an acute or subacute

rheumatic infection. As time progresses there are further changes in the valve as the result of cicatrizing or contracting processes, which may alter the character of the murmur. It must also be remembered that a valve may be so altered by disease that it cannot keep pace in growth with the other parts of the heart.

It is very desirable to be able to differentiate between organic and functional valvular murmurs, but this is not always easy. A mitral regurgitant murmur may be due to other causes than valvulitis, e.g. dilatation of the left ventricle. There are, however, some **characteristics of the organic murmurs** which help in their differentiation. Organic murmurs are usually persistent under varying positions of the patient, during rest and after exercise, and from day to day. They tend in well-marked cases to be louder and harsher and to have a wider range of conduction towards the axilla and back. Yet in the early stages of acute valvulitis the murmur is usually very soft and blowing. Organic murmurs have often a musical element or other characteristic tone, which is not heard in the case of functional murmurs. One or other sound of the heart is often obscure or faint or entirely replaced by the murmur. A definite point of maximum intensity, about the apex or the aortic region, is more marked in the case of organic murmurs. While a systolic murmur may be functional in origin it is a safe rule to regard a murmur occurring during the period of diastole as being of organic origin. Practice will enable one to say in

the greater number of cases whether a murmur is functional or organic. In other cases it may be advisable to leave the murmur out of account and to consider the other symptoms of disease which may be present. In no case is it justifiable to diagnose valvulitis on the evidence of a doubtful mitral systolic murmur.

Valvular murmurs in childhood are associated chiefly with the mitral and aortic orifices, lesions of the pulmonary and tricuspid valves being extremely rare in connexion with acquired disease. The commonest is the **systolic murmur of mitral regurgitation.** In cases of rheumatic fever it is valuable evidence of the occurrence of valvulitis. The murmur may arise during the acute infection or may only become audible at some later period. It may last for a time and then disappear entirely, or it may become permanent. The systolic murmur of mitral regurgitation is a very common sign in early life, and may be the result of a functional disturbance or of organic (valvular) disease. In neither case does it appear to me to merit the importance which is so often attached to it. In the majority of the cases due to valvulitis the murmur is the result of a slight reflux at the valve which does not seriously affect the functional power of the left ventricle. We do not usually find that there is much dilatation or hypertrophy of the left ventricle. The so-called ' compensation ' is accomplished without any special effort on the part of the myocardium. If there should occur signs of cardiac failure it will usually

be found that these are due to some lesion or lesions other than the mitral reflux, and that the systolic mitral murmur shows no alteration during the period of cardiac breakdown.

A diastolic mitral murmur is clear evidence of valvular disease. It is not present in the early stages of valvulitis as a rule, but results from changes in the valve of a cicatricial nature and leading to obstruction. Clinically we find this murmur usually in cases of pronounced carditis, in association with a mitral systolic murmur, and with symptoms of cardiac weakness. The presence of this double mitral murmur may be taken as suggesting strongly not only that the valve has been seriously involved in the inflammatory process, but also that changes in the myocardium have accompanied such valvular disease. Signs of some limitation in the cardiac functions will usually be found.

The typical crescendo murmur of mitral stenosis, **the presystolic murmur,** is not as a rule well developed until after the age of ten or twelve years. Nevertheless, at an earlier age one may hear a prolongation of the first sound terminating in a sharply accentuated manner which is strongly suggestive of commencing stenosis at the mitral area. Herein lies one of the indications of the small value of murmurs *per se*, for the one which of all others is later in life indicative of serious obstruction is not present in childhood. On the other hand, the lesion which causes the murmur arises in the great majority of cases during childhood. The lesion is rheumatic

endocarditis affecting the mitral valve, and producing adhesion of the two segments. As the years pass there is an increasing narrowing of the orifice from fibrous contraction of the valve, and also a relative narrowing of the orifice from want of development *pari passu* with the rest of the heart. A typical presystolic murmur never develops during an acute attack of rheumatic fever. If it is found during an acute attack, then its presence is clear evidence of rheumatic valvulitis at some previous time. In the routine examination of older children and adolescents apparently healthy, one will frequently discover a definite presystolic murmur. Some will give a history of rheumatic fever, others of rheumatic pains, others of chorea only, and others of no previous illness suggestive of rheumatic infection. We know now that the significance of such a presystolic murmur, even in one apparently healthy, is grave because of the cardiac embarrassment which will almost invariably follow. In young subjects there is great variation in the character of the presystolic murmur from time to time, and under varying conditions. It may be diastolic in time, or mid-diastolic, or presystolic. It may change in its time of occurrence from changes of position in the patient (sitting up or lying down), from increased heart rate (as under excitement), and from increased heart effort (exercise). The variations may probably be traced to two factors: first, if the ventricular suction at the outset of diastole is stronger than the auricular pressure, we may expect the murmur

chiefly during early diastole; and secondly, if these conditions are reversed, we may expect the murmur to be presystolic in time. The gradual hypertrophy of the left auricle as time goes on establishes the presystolic rumble as the more permanent condition.

An aortic systolic murmur is not common in early life. It may be due to congenital defect in the valve or in the aorta itself. The determination of the exact lesion giving rise to the obstruction is always difficult, and one would hesitate to diagnose valvulitis in the absence of signs of endocarditis at other valves. The importance of the murmur is probably slight in the absence of any evidence of hypertrophy of the left ventricle as the result of aortic obstruction. **A diastolic murmur produced at the aortic orifice** is always due to valvulitis. It is not uncommon in the more serious forms of carditis, and is always associated with signs of mitral disease. The murmur is to be recognized by a peculiar tone, quite different from that of a mitral diastolic murmur, and by its area of conduction, which is almost always along the left border of the sternum. Its maximum intensity is usually close to the sternum, on the left side, in the 3rd and 4th intercostal spaces, and it may be quite audible down to the ensiform cartilage. On the other hand, it is frequently inaudible or only faintly heard in the so-called aortic area. An aortic diastolic murmur is the result of late cicatricial changes in the valve and consequently does not usually develop during an attack of rheumatic fever. I have found it present some time after an attack

when there had been no sign of aortic involvement during the acute illness. It is to be considered, as a rule, as an evidence of serious endocardial involvement, but, on the other hand, it may lead to no cardiac disability and may even ultimately disappear. Its importance is not to be estimated by the loudness of the murmur, but by the evidence supplied as to the amount of blood which is returned to the ventricle, and the response of the ventricle to the extra call on its powers. If the amount of blood regurgitated is considerable the ventricle, when the myocardium is sound, will respond by dilatation and hypertrophy. Hence comes one of the forms of large heart in childhood. When one finds a heaving præcordium and a powerful shock on palpation, along with evidence of great cardiac enlargement, the two commonest causes to bear in mind are adherent pericardium and aortic regurgitation. The signs associated with the latter in adult life, namely, collapsing pulse, reddish flush about the face and ears, and pulsating vessels may all be reproduced in early life.

The **significance of organic valvular murmurs**, taken by themselves, is very little and should not be exaggerated. We may assume that such a murmur is to be regarded as an indication of a lesion of a valve, and that it is the valvular lesion which has to be considered and not the murmur. But even the valvular lesion may not declare its significance readily. One may express it thus, that a valvular lesion speaks to us through an audible murmur,

communicates its presence, but does nothing more. As regards the future of the lesion, its persistence or disappearance, its effect on the functional activity of the right heart or the left heart, and its association with other cardiac lesions, the murmur gives no information. So that in seeking to determine the significance of a valvular lesion, as evidenced by a murmur, we have to consider what effects, if any, the lesion produces on the heart and circulation. These will be found in some cases in the form of dilatation, hypertrophy, cyanosis, breathlessness, &c. Even in the presence of such symptoms care must be taken lest in ascribing the symptoms to a valvular lesion one has overlooked associated changes in the myocardium, which may be much more responsible than the valvular lesion. In other cases we shall find that there are no signs or symptoms referable to the valvular lesion. We have, then, no means of determining exactly its significance, and it is therefore advisable to hold our hand as regards prognosis and treatment. 'Never prophesy unless you know' is a very good rule to bear in mind in connexion with valvular murmurs, and as they give us very little knowledge as to the future prospects of the patient, cardiac or otherwise, the best plan is not to prophesy at all on a basis of cardiac murmurs.

CHAPTER XVIII

PERICARDIAL MURMURS

The occurrence of pericarditis in connexion with rheumatic infection is much more common in childhood than in later years. The chief sign by which it is recognized is the **friction murmur**, pain being sometimes present and sometimes absent. Pericardial friction may develop during an attack of acute rheumatism, or in the course of chronic carditis. It usually commences at the base of the heart, and may be heard nowhere else. In other cases it extends along the sternum, and may spread over the whole præcordium. The murmur in its typical form is of a to-and-fro character, corresponding to the systole and diastole of the heart; in some cases it is systolic in time only. For some reason which I do not quite understand, pericardial friction in the young is very frequently overlooked. The sound is different from the valvular murmur, being soft and rubbing in acute cases, coarse and grating in chronic cases. It is more superficial than the valvular murmur, and is often markedly intensified by pressure with the stethoscope. If effusion takes place into the pericardium, the friction murmur disappears.

The recognition of a pericardial murmur is of great importance, because it is always indicative of organic disease. In a first attack of rheumatic fever

and in ordinary subacute attacks, pericarditis is not common. But in the case of two children of the same age, namely, four years, I have found acute pericarditis in what was apparently the first attack of rheumatic infection. Probably the cause was the extreme severity of the infection, for other evidences of general carditis were present, there was great dilatation, and a fatal termination soon followed from cardiac failure. The condition which I have been led to associate chiefly with pericarditis is that of chronic carditis or chronic infection of the heart. The typical associated signs are a continuously rapid action of the heart, progressive dilatation, irregular attacks of pyrexia, and pleuritic friction. Chorea in its acuter forms is often accompanied by pericarditis. In all of these cases the significance of the pericarditis lies in what it suggests, viz. that the heart has already been very seriously attacked by rheumatic infection, and of this confirmatory signs are usually present. On the other hand, we may find evidences of adherent pericardium at a late stage without any previous history of acute illness sufficient to entail rest in bed. One can only conclude that in such cases the pericarditis had preceded any extensive affection of the myocardium or endocardium.

CHAPTER XIX

CIRCULATORY DISTURBANCES FROM CARDIAC DISEASE

THERE are various effects manifested in the system as the result of failure on the part of the heart to carry on the circulation in an efficient manner. The most prominent are cyanosis or pallor, œdema of the extremities and ascites, pulmonary engorgement, enlargement of the liver, and oliguria. All of these are of common occurrence in connexion with heart disease in adult life. In childhood, on the other hand, such evidences of cardiac failure are much less common amongst cases of heart disease. This is because the functional activity of the heart has not been impaired. The seeds of disease may be scattered through the heart, and may be destined to produce their dire effects in later years, but their influence on the heart meantime is not such as to induce functional impairment. So that the absence of such signs is no warrant for the conclusion that the heart has not been affected, temporarily or permanently.

The **mechanism** by which these **signs of cardiac failure are produced** is undoubtedly the same in early as in later life. In former years, as a valvular lesion was commonly present in cardiac cases it had been usual to assign the breakdown of the heart to the valvular lesion. But as there is often no change whatever in the valvular lesion it has become

increasingly difficult to explain the sudden or slow development of circulatory disturbance in this way. The myocardium has now come to occupy a more important place in connexion with cardiac failure. The myocardium generally is a complicated subject, and clinically I think one may say that the important factor in the maintenance of a healthy circulation is the **driving power of the left ventricle.** It will also be found that the leading feature of cardiac failure in all its forms is the weakened functional activity of the left ventricle. This may be described as axiomatic, but its application in medicine has not been generally accepted.

Under conditions of health the left ventricle has to maintain as a minimum a flow of blood and a pressure of blood sufficient for the needs of the organism. It has aids in the form of the arteries, the auricles, the right ventricle, and the respiratory movements. But if one or other of these becomes defective or gives out, the necessary compensation will ultimately fall on the left ventricle. *In the case of valvular disease, of myocardial disease, of a disordered rhythm, or of adherent pericardium, the chief questions to be settled are always, how does it affect the action of the left ventricle, and how does the left ventricle respond to meet the difficulty?*

While the majority of cardiac cases in childhood and youth do not reach the stage in which evidences of circulatory disturbance are present, there remains a large number in which those signs of cardiac failure are manifested. As a rule they are an

evidence of progressive carditis, the result of repeated outbreaks of rheumatic infection. Their appearance is even more significant than in adult life, for the course towards a fatal termination tends to be more rapid. In another class of case the circulatory disturbances may be the result of a disordered rhythm, and if the myocardium is healthy, the embarrassment of the left ventricle may be only temporary. In another class we may find the circulatory disturbance develops as the result of an acute illness, e. g. influenza, when the heart had been previously damaged. When the effects of the acute infection have passed off, the functional activity of the heart may return to its previous condition.

In cases of **acute heart failure** signs of circulatory disturbance like œdema are not commonly met with or only in a very minor degree. Œdema may be present before death from acute carditis. What one does find is a marked enlargement of the liver with cyanosis. The liver seems to become engorged with blood on the slightest failure in strength of the left ventricle. Even in **chronic cardiac cases**, while œdema of the extremities and ascites may occur, it is by no means as common as in adult life. It would appear to be a characteristic of long-standing cases of heart disease rather than an acute manifestation. The characteristic sign in childhood is enlargement of the liver, and this should always be watched for as one of the earliest signs of a failing heart. Pulmonary congestion and even hæmoptysis are not uncommon. Diminution in the amount of

urine and the presence of albumin and tube-casts may also occur as the result of cardiac failure. The significance of the signs of circulatory disturbance is undoubtedly great, because when persistent they indicate an advanced stage of heart disease, of which other signs will be found on examination of the heart.

CHAPTER XX

SUBJECTIVE SYMPTOMS DUE TO CARDIAC DISEASE

There are certain symptoms of discomfort complained of by patients suffering from cardiac weakness, the significance of which is very great in adult life. Of these the chief are faintness and fainting, palpitation, shortness of breath, præcordial pain or discomfort, and mental anxiety. These do not possess the same value in childhood for various reasons.

In the first place, we do not find that children observe and dwell on those symptoms as their elders do, and so we cannot get a very explicit or intelligible statement of their character.

In the second place, they are not nearly so commonly met with, and their absence is no evidence as to the patient's freedom from heart disease. I have emphasized this point before, that in childhood these subjective phenomena are entirely absent

in the vast majority of cardiac cases. They are absent in many cases in which the physical signs about the heart are conclusive evidence of the presence of carditis, which may be in an active or a quiescent stage, and in an early or an advanced stage.

In the third place, the symptoms are quite common amongst patients who have no cardiac disease. This has already been referred to in connexion with the subject of cardiac disturbance from nervous or vasomotor instability. Patients of this class are extremely liable to such symptoms, and possessing, as they usually do, a remarkable sensitiveness to abnormal sensations, they are by no means silent on the subject. Hence a complaint of fainting or palpitation in childhood or youth is much more suggestive of nervous disturbance than of cardiac disease.

In the fourth place, the onset of these symptoms due to cardiac disease is usually very gradual. We are not accustomed in the case of children to meet with the sudden onset of symptoms of cardiac failure such as is so common in adult life. The gradual development of such symptoms does not attract the notice of a child, still at an unobservant and unreasoning age, and still unexposed to the more severe strains on the heart which are common in adults of both sexes.

For these reasons we do not place much reliance on the absence of a history of these subjective phenomena in any given case. If they have not been complained of by the patient, we may get

some information by questioning the parents as to the presence of any symptoms pointing to cardiac distress, and by giving the child such active exercise as will elicit them under examination.

The general public has very clearly defined views as to the symptomatology of heart disease, and children will be suspected of some cardiac trouble should one or other of these symptoms develop. In this connexion it must be kept in mind that disease or disturbance in some other part of the body may cause the symptom, and is in fact much more likely to have done so. Because a child has fainted, or because a child has become short of breath on running, it is not justifiable to diagnose heart disease and to commence a course of cardiac treatment. I think it may be laid down as a safe guide that if a child has cardiac symptoms there will also be objective signs of heart disease on physical examination. Further, a definite co-relation must be established between the signs which are found on physical examination of the heart and the symptom or symptoms which have led to the examination. The detection of a systolic murmur at the apex is not to be regarded as in itself a sufficient explanation of fainting or palpitation or breathlessness in the child. The presence of cardiac symptoms marks a late and not an early stage of organic heart disease, and if the symptoms precede the physical signs one ought to look for some cause apart from the heart for their origin. For example, fluid effusion in the pleura may lead to shortness of

breath, flatulent distension of the abdomen may cause palpitation, anæmia may lead to fainting, pain about the præcordia may be due to muscular rheumatism, and so on. If an examination for an extraneous cause of 'cardiac symptoms' were always made in a systematic manner, the heart would often be freed from unjust suspicion, and the patient would often be saved from uncalled-for treatment.

The development and progress of these subjective phenomena of cardiac distress will, however, be met with in certain cases during childhood. These cases are the ones in which the carditis is progressive, or in which extensive cardiac disease is present. We then find the same phenomena as are met with in adult life in connexion with cardiac failure, and as a rule the course towards a fatal termination is more rapid during early life. Some are of greater and some of less significance. Sometimes they are elicited only when the patient is putting extra strain on the heart, as in walking or running; in other cases they are present even when the patient is lying quietly in bed. Sometimes they may be associated with an acute cardiac lesion, and in other cases they may be merely an indication of cardiac failure from old-standing disease. In one particular they differ from the physical signs of heart disease which have been described in that these phenomena invariably indicate a condition of the heart which calls for treatment.

Faintness and **fainting** may be associated with aortic regurgitation, as in adult life, or with cardiac

dilatation. It is usually dependent on a defective supply of arterial blood to the brain. It will naturally be more liable to occur under conditions of over-exertion or excitement.

Shortness of breath and **dyspnœa** are somewhat complicated in their origin, and the exact origin of cardiac dyspnœa is not yet clearly understood. In many cases there would appear to be a disturbance of the respiratory centre in the medulla, causing a condition of air hunger. This is probably induced either by a deficient supply of arterial blood or by an altered condition of the blood. In other cases there is a defective circulation in the lungs and imperfect aeration of the blood leads to dyspnœa. In a third class the breathlessness is associated with anæmia of a marked character, the result of the rheumatic infection. Dyspnœa is often a striking symptom in connexion with acute pericarditis and the later stages of adherent pericardium. The sudden onset of dyspnœa should always lead to a careful examination for signs of pericarditis. Shortness of breath in heart cases is always indicative of serious cardiac embarrassment, and in chronic cases it marks a stage in the downward progress. In the more severe cases orthopnœa may be permanently established, as in adult life.

Pain in the cardiac region may be associated with pericarditis, and is often the first symptom to attract attention. On the other hand, an attack of pericarditis may be passed through without the complaint of any pain on the part of the patient. It is

difficult in the case of young people, and especially those acutely ill, to make investigations as to the character and exact seat of pain. In acute pericarditis the pain may be over the whole præcordial region and associated with surface tenderness. Or it may be referred to the neck, the left shoulder, or the left arm. The question as to the exact nature of the pain in pericarditis is a large one, but probably two forms are present, and have to be considered. One is the sharp pain of pericarditis, comparable to that in acute pleurisy. The other is the expression of the labouring heart, the cry of distress from an over-strained and exhausted musculature of the heart. For acute pericarditis is often but one part of an acute general carditis in which the myocardium is in a state of acute inflammation.

In chronic cases pain is often associated with the later stages of adherent pericardium. This may be due to exhaustion of the left ventricle, or to the presence of adhesions causing overaction of some part of the heart. Pain is by no means so common in children as in adults, and in the former we do not meet with the attacks of angina pectoris which are so characteristic of cardiac degeneration in later life. Along with other symptoms of cardiac failure, pain may be induced by any extra strain thrown on the heart, as under excitement or exercise.

Palpitation does not attract the same amount of attention in youthful patients as in adults, nor cause the same amount of anxiety to the patient. It may be felt when there is any sudden increase

of rate in the heart, from acute disease, or exercise, or excitement.

It is only natural that these subjective phenomena should not be so prominent in childhood where previous experience of health and full activity has not established a known standard. It is equally natural that they should become more noticeable in youth when school life and games lead to much more severe strain and excitement. There is one subjective sensation which is usually present even in the early stages of heart failure in childhood, and that is a **feeling of tiredness**. It is manifested by the general languor and disinclination for exertion which are in such marked contrast to the active habits of a child with a healthy heart, and, it may be added, the child more especially with a tendency to rheumatism. When progressive this languor may be regarded as significant of increasing cardiac debility. On the other hand, when it has passed off one may assume that the functional activity of the heart has improved.

CHAPTER XXI

PROGNOSIS

The exact data on which a definite prognosis regarding an attack of carditis could be given are not yet at our disposal. There still remains a great deal unknown and possibly unknowable. So far as

cardiac signs and symptoms are concerned, much and serious damage to the heart may be entirely latent for a considerable time. It becomes all the more important to be able to estimate the symptoms and signs which are present at their proper value, and not to be led astray by any preconceived ideas or erroneous assumptions.

In a first attack of rheumatic infection the heart is not usually seriously affected, provided that proper treatment is carried out. With each succeeding attack the tendency to more serious involvement of the heart becomes greater. Hence, if the patient has had one attack of rheumatic fever with slight carditis, the prognosis may be said to depend chiefly on the possibility of preventing a fresh infection. The power of recovery in youthful hearts is great, much greater than in adult life, but the prospects of a relapse or reinfection are also great, much greater than in adult life. With reference to some children one might say once rheumatic, always rheumatic. While this tendency to fresh attacks of infection lasts, which is right on through adolescence, we cannot give a prognosis as to the future of any damaged heart. We can, however, often say with confidence that the carditis has been so slight that no permanent damage has been done.

In all the relapsing cases of rheumatic infection, the tendency to further and more serious heart disease is markedly increased. In the condition of chronic rheumatic infection there is usually present progressive carditis. Certain signs are of some value

as aids in the prognosis. I have already emphasized the importance of a **continuously rapid pulse** in cases of subacute heart disease. Whether this is the result of a chronic rheumatic toxæmia stimulating the heart, or of definite myocardial changes increasing the heart's excitability, I am not prepared to say. The persistence of this rapid pulse must always be regarded as of grave prognostic significance, and other manifestations of heart disease will always be found to exist. In early life the value of **subcutaneous fibrous nodules** as an aid in the prognosis should not be overlooked. It was held formerly that they were always associated with grave cardiac lesions and that death from heart disease could be foretold within a year. This, however, is too extreme a statement. Their value appears to me to lie in their frequent association with chronic or relapsing carditis. They are a visible and manifest sign of an active rheumatic infection, which we may suspect in connexion with the heart but have not positive proof of. Their disappearance has often seemed to me to be an indication that the infection has also for the time died down, a change which will probably be manifested in other ways also. The appearance and the disappearance of rheumatic nodules has thus a value in regard to prognosis.

While too much stress must not be laid on individual signs, there are some which directly affect the prognosis. **Dilatation of the heart**, with a weak action, i.e. without any indication of hypertrophy, must be carefully watched, and the persistence of

this condition is of serious prognostic significance. Definite **cardiac hypertrophy**, the result of adherent pericardium, will usually lead up to cardiac failure before adult life is reached. **Aortic regurgitation** so marked as to lead to progressive dilatation and hypertrophy of the left ventricle suggests cardiac failure in the not far distant future. **Incipient mitral stenosis** is to be viewed gravely. On the other hand, mitral regurgitation as a single lesion is of little prognostic value.

Other points in connexion with prognosis will be considered in the remarks on treatment following.

CHAPTER XXII

TREATMENT

Considering the serious results which follow from organic heart disease, the desirability of preventing its occurrence is evident. As heart disease is usually secondary to rheumatic infection it is to the latter primarily that attention must be directed. Other forms of acute infection may attack the heart, but in the vast majority of cases in early life it is rheumatic infection that paves the way to heart disease. Thus the more often that the early symptoms of rheumatic infection are detected in childhood, and the more thoroughly they are treated, the smaller will be the incidence of organic heart disease. Unfortunately, in the case of a large part of the

population those early signs are overlooked and medical advice is not sought until the infection has taken a firm hold of the system, and possibly heart disease is already well marked. It is for this class of the population, the young sufferers from early signs of rheumatic infection, that a sanatorium would prove of the greatest value. At present we are building these everywhere for the subjects of tuberculous infection when the disease has taken a firm hold and its eradication is problematical. If one considers the number of adults who are permanently crippled by heart disease acquired in early life, it is clear that if this heart disease is prevented the gain to the community would be immense. That it is preventable is shown by the results of the treatment of rheumatic infection amongst the well-to-do. I believe that if young rheumatic subjects could be removed from their unhealthy home surroundings at an early stage, before the onset of carditis, or when the carditis was but slight, and kept in a sanatorium for one or two years, and efficiently treated there, we should probably succeed in preventing a large amount of heart disease which would otherwise inevitably develop. It must be remembered that we possess a means of treatment for those early cases which is both curative of the infection and preventive of serious heart disease.

In the case of an acute attack of rheumatic infection (rheumatic fever) the possibility of carditis must always be kept in mind. In order to prevent this complication the salicylate treatment should be

thoroughly carried out, and rest during the convalescent stage should be sufficiently prolonged. If signs of carditis appear, which they usually do in the form of a mitral systolic murmur and slight cardiac dilatation, the period of rest must be more prolonged and the treatment by means of salicylate of soda repeated in systematic courses. It has appeared to me that the thorough treatment of a first attack is more effective than the treatment of any subsequent one. Consequently, if there are definite signs of cardiac involvement and persistent signs of cardiac irritability and disturbance (rapid pulse), I should not hesitate in advising a very prolonged rest in bed, for six months or longer if necessary, in the hope of rendering the heart functionally sound. The persistence of a mitral murmur or slight dilatation does not preclude the possibility of securing this result. After the patient has been up and about one will naturally order all precautions to be taken against the risk of fresh rheumatic infection. In addition, from the preventive point of view, it is advisable to give a course of salicylate of soda once a month for one or more years. For a child of ten years twenty grains of salicylate of soda may be taken daily for a week or ten days every month. Tonsils, teeth, and any other possible source of infection should be attended to, and the general health maintained at as high a level as possible.

I emphasize the importance of these preventive measures because of the small effect of any direct

treatment of carditis when well marked and progressive. It is often said that the salicylates are useless in the treatment of carditis, but this is going too far. As regards the direct effect of salicylates on myocardial or valvular inflammation, the result may not be appreciable or manifest. But we have to consider the general rheumatic infection which has started the carditis and which tends to keep it up, and if we can keep this infection under or remove it from the system by means of the salicylates, we are undoubtedly improving the prospects of the patient as regards his heart. This is the effect which the salicylates have, and therefore their use is of great benefit.

When in the course of an acute or subacute attack of rheumatism the heart has become affected, as shown by the signs of valvulitis or myocarditis, the means at our disposal for direct treatment of the cardiac condition are extremely limited. It would be well if this fact were more generally recognized. The natural tendency in the medical mind is to attack any abnormal sign or symptom, and more especially to indulge freely in the use of the so-called cardiac tonics. If we consider the underlying pathological condition in carditis, namely, inflammation of a valve, or of the myocardium, or of the pericardium, or of two or more of these, it is difficult to see how drugs are going to act on these lesions directly. It is often said that if the pulse is weak we must stimulate the heart. But an inflamed heart is more in need of rest than stimulation. If a patient

were suffering from an inflamed muscle in any other part of the body we should at once seek to secure as much rest for it as possible, and not stimulation. The cardiac stimulants, as they are called, form a large class and embrace amongst others brandy, ether, digitalis, strychnine, and caffeine. Now if any evidence were at our disposal that one or other of these medicaments was of real value in active carditis, its use would be justifiable, but at present this is wanting. On the other hand, they may do harm, especially if given in full doses, not probably from any direct effect on the heart, but through their effect on the nervous tissues. For these reasons I think that the less stimulation of the heart that is attempted during the stage of acute carditis, the better for the patient.

We have to consider the effect of **local treatment in carditis**, which may take the form of painting with iodine, blistering, leeching, or hot applications. As regards acute valvulitis and acute myocarditis, it is difficult to see how either can be benefited by such measures, and they have fallen into disuse. When pain is present, which is most commonly associated with pericarditis, I think this local treatment has a beneficial effect—as regards the pain. By some an ice-bag over the præcordium is considered to be valuable not only in relieving the pain of pericarditis but in improving the cardiac conditions generally. Dr. Lees recommends this treatment strongly. From the patient's point of view, I believe that hot applications are more agreeable,

and they are probably as effective. Leeching and blistering are also efficacious, but the former usually excites alarm in the case of children under ten years of age. In so far as we are dealing with a symptom—pain—the local treatment may be beneficial. But when we speak of treating an acute infective inflammation like endocarditis, or myocarditis, or pericarditis by these local measures, and of good results to be obtained, I can only regard this as the triumph of hope over experience. Mild cases recover with or without such local treatment, and severe cases do not recover in spite of such treatment.

It may be said that on these lines the treatment of active carditis in the young becomes rather a negative quality. But I have been dealing so far with what an exuberant therapeutic zeal may lead one to do, and which may not prove in the end to be beneficial. A very great deal can be done in the way of securing as much rest to the heart as possible, which of course means an easy, steady, unhampered action. If we cannot reach directly the sources of disturbance which lie inside the heart, we can at least reach those which lie outside. By securing physical rest and sleep, and by removing all nervous disturbances and reflex causes of disturbance, we can do a very great deal to secure the maximum of rest to the heart. The wise use of the bromides, of morphia, of calomel, and of a carefully regulated diet will do much in the way of attaining these objects. It is in this direction during the stage of acute carditis that we should direct our energies,

and it is probably in this manner that we shall do most to benefit, and least to harm the patient.

In like manner, after the acute stage of the rheumatic infection has subsided, the question of prolonged rest to the heart may be the important consideration. We may find that a certain degree of pyrexia persists, as shown by an evening rise of temperature. This may be due to the persistence of the rheumatic infection and calls for further treatment by salicylate of soda. On the other hand, in the case of children it may be associated with prolonged rest in bed, and this can be tested by getting the patient on to a chair for a few hours daily and watching the effect. If the temperature ceases to rise we may conclude that the active general infection has gone. As regards the heart, it is desirable to maintain the absolute rest in bed as long as any signs of active carditis persist. Of these the most useful are an increased heart rate and dilatation of the left ventricle. The most satisfactory sign of recovery after acute carditis is a slowing of the pulse rate to 70 or 80. If the rate continue above 100, and no other cause can be found, we may assume that carditis is still present and further rest is required. Dilatation of the heart is easily induced in early life, and too much stress must not be laid on a slight degree of it. If, however, there has been definite dilatation taking place during the acute attack, it is very desirable to secure a return to normal conditions before allowing the patient out of bed. The presence of a systolic murmur is not

of so much importance. If there has been definite valvulitis the murmur will usually persist for some time if not permanently. In the case of children one often finds that the murmur disappears after, it may be, one or two years. This applies to the ordinary mitral regurgitant murmur and less frequently to the aortic regurgitant murmur. Under any circumstances the prolonged rest treatment, merely because of the persistence of a systolic murmur, will not affect the valvular condition and may adversely affect the child's general health.

The convalescent stage, when the patient is allowed out of bed, must be conducted in a tentative manner. The home surroundings should still be of a quiet character, and all nervous excitements should be carefully avoided. At first the change should only be from bed to sofa or an easy chair. By degrees a little walking about the room may be permitted, and after a long rest this will certainly try the legs as well as the heart, so that one must not ascribe the weakness necessarily to the heart. Careful observations must be made as to the effect of exercise on the heart, and more especially to the presence of breathlessness or fatigue on walking. The amount of liberty that is to follow must be determined by the presence or absence of signs of cardiac weakness or irritability. After an attack of rheumatic infection with carditis it is advisable that a period of at least six months should elapse before a child returns to ordinary pursuits and school life. In most cases the future prospect as regards

the heart depends chiefly on whether or not a fresh rheumatic infection takes place.

The various forms of **chronic heart disease** in early life call for careful discrimination in the way of treatment. Reference has already been made to the custom in the past of diagnosing 'a weak heart' or 'an irregular heart', or 'a cardiac murmur', and then laying down such restrictions in the normal habits of childhood as really meant a state of chronic invalidism. The tendency was to regard anything out of the normal as a danger signal. Erroneous physiological standards of cardiac sounds and action were drawn up, and anything outside was considered to be pathological and of grave significance. Present-day methods are more exact and more rational. We seek to analyse the source and character of an abnormal sign or symptom; we try to distinguish that which is of importance from that which is not; and we do not adopt any treatment unless there is some clear indication that such treatment is called for. No doubt the position taken up by the cautious therapeutist who says that heart disease may develop if his restrictions are not carried out is a strong one, for it cannot be disproved. But it is not a position based on any scientific data, as a rule, and when the diagnosis is founded on a general finding such as that mentioned above, it is absolutely wrong. In early life there is either heart disease or there is not, but in the latter case many disturbances due to extra-cardiac influences have in the past been put down to heart disease. One may be in doubt

for a time, and the patient may have to be carefully observed, but it should always be possible by this means to say whether heart disease is present or not. Treatment, whether active by means of drugs, &c., or passive by means of restrictions in the ordinary routine of life, should not be adopted until this question has been settled.

In these days, when the whole of the youthful population comes under medical examination, cardiac irregularities and abnormal sounds are discovered much more frequently than formerly. The family doctor is constantly confronted with the problem of deciding as to the significance of any such sign, and has to answer the question, 'Has the child got heart disease?' The lay mind dwells largely on heart disease, and the occurrence of faintness or fainting, breathlessness, or chest pain will often lead to the seeking of medical advice, the diagnosis of a cardiac origin having been made already in the family circle. These problems can only be satisfactorily dealt with if one has a clear grasp of the real distinctions between normal action, functional disturbance, and organic disease of the heart. We may recapitulate here some of these distinctions which have been dealt with in former pages.

1. Irregularities of action are comparatively rare from organic disease in early life; they are very common from nervous disturbance.

2. A systolic murmur heard in the præcordial region is not evidence of heart disease, and, taken by itself, is never an indication for treatment.

3. The so-called symptoms of heart disease are rarely met with in early life except as the result of advanced disease; similar symptoms are common as the result of nervous disturbance.

4. Acquired heart disease apart from evidence of rheumatic infection is extremely rare.

The subjects of chronic heart disease present themselves for treatment under varying conditions of disease. One large and important class of sufferers is that in which **recurrent attacks of rheumatism** are taking place. These are apt to be associated with fresh attacks of carditis, mild possibly, but always tending to leave the heart more crippled. The essential part of the treatment here is to combat the rheumatic infection by salicylates, change of residence to a warmer and drier climate, &c.

In another class **progressive carditis** is present; that is to say, the rheumatic infection has settled on the heart and established itself there. A condition of chronic myocarditis is present and the valves and pericardium may also be involved. Frequently there are no subjective symptoms complained of, and the patient can be up and about without any personal discomfort, although the general health is manifestly below normal. The temperature may be slightly pyrexial at times, or it may be quite normal. Other signs of rheumatic infection may be present and more especially are fibrous nodules about the tendon sheaths and periosteum associated with this condition. On examination of the heart the chief signs of importance are a persistently rapid action

and a tendency to progressive dilatation. In such a condition one naturally turns to the use of salicylates, to eradicate the rheumatic infection, but unfortunately the result is seldom satisfactory. We seem to have reached a stage of local rheumatic infection as opposed to the blood infection of rheumatic fever, and the satisfactory results obtained in the latter cannot be reproduced in the former. Nevertheless it seems to be the right thing to do to give a full course of salicylates, and to repeat this at intervals. I have sometimes wondered in these cases as to whether the rheumatic infection had implanted itself on some other tissue and was from that stronghold pouring out the poison into the system. With this idea tonsils, teeth, kidneys, and alimentary tract have all been examined and put into as healthy a state as possible, but so far I have not been able to trace any definite results from this line of treatment. It has not appeared to be advisable to give any drugs to lower the heart rate, and far less to give any of the so-called cardiac tonics (digitalis, &c.) in this condition. There are two things to be kept in mind. First, a chronic infection like this tends to die out if one can maintain the normal resistant powers of the body at a high level. Consequently a diet of nourishing food is to be ordered, and a gain in weight will be an indication of satisfactory progress. Secondly, the cardiac condition is most likely to improve under rest and freedom from disturbance. The patient is therefore kept in bed for a prolonged period. Open-

air treatment may prove very beneficial. Until we have at our disposal some better means of combating a chronic rheumatic infection our therapeutic measures in this condition must be limited, but patience in treatment will often be rewarded with a satisfactory result.

The outlook is more hopeful in those cases in which the rheumatic infection has been entirely removed and an interval of perfect freedom has elapsed. We may find evidence of cardiac involvement in the shape of a murmur or dilatation, but no signs of recent or active heart disease. We shall assume that there is no evidence of cardiac disability in the form of breathlessness or cyanosis. From the therapeutic point of view we are dealing with a past lesion of the endocardium or myocardium, and the question is, can we affect this condition in any way? As recovery has taken place it is difficult to see what treatment can possibly affect the cardiac functions which are unimpaired. As regards treatment of the cardiac condition the less that is done the better. As regards future dangers, the chief one to bear in mind is the possibility of a fresh rheumatic infection, and precautionary measures may well be adopted to avoid this complication. The other danger which will present itself to the minds of the relatives and the physician is that of cardiac weakness in the future. I do not think this can always be definitely decided at once in all cases, although in the case of ordinary lesions the ultimate prognosis is very good. If, however, there is mitral regurgitation or

aortic regurgitation sufficient to cause dilatation and hypertrophy of the left ventricle, then in all probability the functional power of the heart will suffer more or less in later years. This, however, is speculating in futures and does not directly affect the immediate treatment. It is also to be noted that the foundation of mitral stenosis may have been laid, but, as in the early stages there is no method of detecting this condition, the question of prognosis and treatment does not arise. The regulation of the patient's life will next come up for discussion, and diet, exercises, games, and school life will have to be settled. The less interference there is with the ordinary pursuits of life in such cases the better for the patient. As a matter of practical experience, it will be found that a child suffering from cardiac weakness will not over-exert himself in an injurious way, provided he is not placed in competitive surroundings. There is nothing to be lost and much to be gained by allowing our patient to resume ordinary habits. If he flags and fails, he will certainly show it by unmistakable signs, which can easily be recognized in the case of a child at home, and further treatment can be employed. In the case of youths at school it is advisable to forbid the excessive strain which accompanies football, running races or paper-chases, rowing, and all athletic competitions, for a time at least. After an attack of carditis the estimation of the functional power of the heart can be made only by testing it in the pursuits of ordinary life. No young heart

can develop to its full power if the owner is, so to speak, wrapped in cotton wool. If we consider the causes of breakdown in the heart disease of adult life, we find that it is usually due either to mental overstrain or physical over-exertion. These causes can easily be prevented in the case of young subjects, and consequently the risks of allowing an ordinary life are very much lessened. For these reasons it appears to me that those cases which present only one or more objective signs of previous carditis, and nothing else, should be started again as healthy individuals, under observation, but not under restrictions, save as mentioned above. Acting on these lines I have found that the majority of cases do well and suffer no harm. A certain number fall back as the result of fresh rheumatic infection which had not been or could not be averted. A small number show signs of progressive cardiac weakness, and in all probability they would have done so under any form of treatment, as it was clearly the result of the original rheumatic carditis. I do not think that those ultra-cautious physicians who, because of a past cardiac lesion, condemn a child to a life of invalidism can show better results. It is no kindness to patients or their relatives to be always hinting at possible evils in the future of which we have no real knowledge. The only test of the functional power of the heart which we possess is the test of experience in the individual. The test is applied by time and by giving the heart some work to do. If the heart does not stand the test we shall

acquire a definite basis of knowledge as regards future treatment, and we shall not have done the patient any harm.

We now pass on to a more serious class of case —that in which symptoms of cardiac failure have appeared. In chronic heart disease this may be brought about by various outside causes, such as acute illness of any kind, and may be of a temporary character. We are considering rather the cases of chronic cardiac disease in which the symptoms of failure are primarily due to weakness of the heart itself. Temporary symptoms may be the result of strain thrown on the right ventricle, and permanent limitation of the cardiac response to exertion may be aided by the same cause, but such conditions do not play a prominent part in early life. Whatever the special lesion may be, and wherever it may be situated, the question of cardiac failure resolves itself into the effect of the lesion on the functional power of the left ventricle. Any treatment which is to be serviceable must be directed to improving the power of the left ventricle.

The symptoms of cardiac failure vary greatly in different cases, but speaking generally they are the same in early as in adult life. The subjective phenomena are breathlessness, tiredness, sleeplessness, and pain. The objective signs are enlarged liver, œdema, ascites, cyanosis, pallor, oliguria, and cardiac enlargement. These symptoms, which vary greatly in different cases, are, as a rule, of more serious prognostic significance in early life because

they point either to progressive disease or to an advanced stage of cardiac weakness. Adult hearts may for a long time show only a limited loss of functional power, and many attacks of cardiac failure are capable of such treatment as will restore the functional capacity to a great extent. The causes of cardiac failure in early life are seldom amenable to such successful treatment. One may note here that the condition of auricular fibrillation which is so successfully combated by means of digitalis is but rarely met with in early life. While in adult years many of the cases of cardiac failure are not associated with any inherent weakness of the left ventricle, in early life it would appear that the myocardium of the left ventricle is especially liable to give way. The onset of signs of cardiac failure must therefore be viewed somewhat gravely, and a marked limitation of the power of the heart to respond to exertion must be regarded as probable in the future. None the less, although the activities of the patient and the working capacity must be curtailed, a very great deal can be done in the way of treatment to build up the cardiac powers.

The first consideration in the way of treatment is to **ease the work of the heart as much as possible.** If the symptoms are only slight, such as breathlessness or tiredness on exertion, and the cardiac condition is satisfactory, it may be sufficient to moderate the amount of exercise and exertion so as to avoid the production of the symptoms. One may find that the disturbance has been caused by the

neglect of some of the simpler rules of life, and when this has been corrected the cardiac action may again become satisfactory.

Complete rest is the chief means of lightening the heart's work when the symptoms are more marked. At first the rest should be in bed, the patient either lying down or propped up as is found to be most comfortable for breathing purposes. Later on the rest can be continued on a couch during the day, either in or out of doors. What must be insisted on is a sufficiently prolonged period of rest, possibly for some months, as the heart will improve under any conditions but slowly, and the less work it has to do the more progress it will make. It is also important to keep away any causes of nervous disturbance, so that the presence of a quiet nurse and the exclusion of visitors will often help to expedite recovery. Sometimes the nervous system of the patient is an excitable one naturally and cannot easily be kept quiet. In such a case the reaction of the nervous system on the cardiac condition may be most injurious, and for this and sleeplessness one should not hesitate to give bromides in full doses. The state of the organs below the diaphragm must be attended to, as flatulent distension of the stomach, or constipation, or an engorged liver, may seriously interfere with the cardiac action. Flatulence will often be best avoided by a diet which is solid rather than sloppy, and which contains fish, fowl, mutton, eggs, and toast as the chief ingredients. The meals should be small in amount, and given more fre-

quently than under normal conditions. Fluids should be taken between rather than at meals. Enlargement of the liver, which is a valuable and common sign of cardiac failure, is to be relieved by mercury and salines. When the signs of congestion of the liver or lungs are present, or when the patient is cyanosed, the value of leeching and of venesection must not be forgotten. If a state of restlessness is caused by præcordial pain or discomfort, it is advisable to give opium in full doses.

In addition to relieving the work of the heart one seeks to **stimulate and strengthen the cardiac muscular tissues.** It must be admitted that in many cases our therapeutic resources in this direction are limited, and it is as well to recognize the fact. In some cases the signs of cardiac failure mark the terminal stage of progressive carditis, and attempts at stimulation of the degenerated tissues have but little effect. More hopeful are those cases in which from some cause there has been a temporary breakdown or weakness of the heart muscle. For immediate results brandy still takes a prominent place in domestic circles, and probably deservedly so. Its effect is to be regarded as stimulating and in no sense strengthening the cardiac powers. It has in addition the result of calming the nervous system and dilating the peripheral arteries, both of which actions may be regarded as beneficial as regards the heart. The direct action of strychnine on the heart muscle is probably but slight, and in consequence its use as a cardiac tonic has been disputed. One

has to remember, however, that the various cardiac functions are controlled by the nervous system and that it may be possible to reach the heart via the nervous system. Clinical experience points to a certain restorative effect of strychnine, given hypodermically or by the mouth, in cases of heart failure, and especially in the course of chronic disease. My own experience has been that young subjects stand strychnine well, and that it has been of service in tiding over a period of heart failure. In cases marked by cyanosis or cardiac dyspnœa oxygen is often useful. There is one great advantage about the use of oxygen, namely, that one can tell very quickly whether it is acting beneficially. If it is found to relieve the cyanosis, to calm the breathing, or to soothe the patient, then it should be persevered with. The oxygen may be administered for from ten to twenty minutes every few hours, according to the state of the patient. It is very difficult to say beforehand whether an individual patient will be benefited by oxygen or not, and often in apparently suitable cases, e.g. those with marked cyanosis, the results are disappointing. Nitro-glycerine and the nitrites are not much called for in the case of youthful patients, in whom vascular spasm and thickening of arteries do not occur as in adult life. In some cases, however, characterized by pallor, cold extremities, and a thready pulse, nitro-glycerine by the mouth or the inhalation of nitrite of amyl will prove beneficial. All of these remedies have only a temporary effect in stimulating the heart

and tiding the patient over a period in which the cardiac symptoms are more or less urgent.

When we come to the **direct cardiac tonics** we find that the digitalis group—including digitalis, strophanthus, and squills—is by far the most efficient. Within recent years, and largely through the work of Dr. James Mackenzie, the real value, and at the same time the limitations of digitalis, have been much more clearly defined. In view of the statement made above, that the chief factor in cardiac failure is weakening of the power of the left ventricle, it would be very satisfactory to have it clearly ascertained whether digitalis does or does not directly increase the power of the ventricular contractions. Physiologists teach that when the ventricle contracts it does so with all the power it possesses at the time, that every contraction in fact is the maximum possible. As this statement is generally accepted it is difficult to see how digitalis can act beneficially on the ventricular wall except by increasing the muscular tone or contractility. Such an action has not yet been shown to exist. When we study the conditions in which digitalis is of proved benefit its action is clearer. The most striking condition is that of auricular fibrillation, in which a damaged ventricle is still further weakened by the disordered cardiac rhythm. In this case the important action of digitalis is to slow the heart, and by the lengthening of the period of ventricular diastole the force of the ventricular systole is greatly increased. We have evidence here of the action of

digitalis in diminishing the excitability and the conductivity of the heart, but we have no evidence of its increasing the contractility of the ventricle. This condition of auricular fibrillation is but rarely met with in early life. Out of 116 cases referred to by Dr. Thomas Lewis, only four were under the age of twenty years. My own experience has been that digitalis will prove equally beneficial in the youthful forms of auricular fibrillation, if given in the proper manner.

It would appear that the most striking results from digitalis are to be obtained in cases of disordered rhythm in which a slower action of the heart is desirable. Further, this slowing action seems to follow from a direct stimulation of the vagus nerve, and the effect is recognizable on every chamber of the heart. If the cardiac rate is normal or if the rhythm is quite regular, beneficial results from digitalis are not so apparent, in fact they are usually wanting. These facts, which are now generally accepted, would seem to show that any direct action of digitalis on the muscular tissues of the heart has not so far been proved to exist. They also tend to explain why the range of digitalis therapeutics in early life is much more limited than in later years. In the young disorders of rhythm in the form of irregularities are much less common in association with organic cardiac disease. An increase of rate may often be met with, but in the majority of cases this is due to carditis, either of a toxic or an inflammatory nature, and it is well known that the action

of digitalis in slowing the heart is not effective under such conditions. There remains, therefore, but a comparatively small number of cases in which digitalis may be hopefully used.

These cases are the ones in which exhaustion of the left ventricle has come on, in the absence of acute disease, and as the result of over-exertion or some other strain. The main clinical indications will be as follows : (1) dilatation of the left ventricle, (2) rapid cardiac action (100 to 150), (3) œdema of the extremities and ascites, (4) diminution in the amount of urine, (5) breathlessness. When one or more of these indications of cardiac failure are present digitalis should always be tried, even although its mode of action may not be clear.

The action of digitalis, as ordinarily administered, is not manifested for some days, and it is not therefore of any use in emergencies, when more rapid stimulants are called for. It is very important to push the drug until some evidence of its action has been obtained, and individuals vary greatly as to their susceptibility to digitalis. As a rule young people take digitalis well and often tolerate with benefit larger doses than are usually given. If one employs two or three minims of the tincture thrice daily it may be weeks before any effect is produced, and the practitioner may discard the drug as useless for his patient, whereas he has only been giving too small doses. For a child of twelve years the initial daily amount should be from thirty to forty drops of the tincture. A careful record should be

kept of the pulse rate, the amount of urine, and the presence or absence of vomiting. When the pulse begins to be steadily lowered, or when the amount of urine is increasing, the dose of digitalis may be reduced one-half. Vomiting arising during the administration of digitalis is also an indication for intermitting the treatment or diminishing the dose. When the cardiac rate is normal and there is no œdema there is not the same opportunity of seeing the immediate results of its action. It should be administered in smaller doses, and the development of any marked slowing of the pulse, or irregularity, or a coupled rhythm should be regarded as an indication for intermitting the digitalis.

CHAPTER XXIII

TREATMENT (*continued*)

Special Types of Disease

There are certain types of carditis in which the prognosis is very unfavourable, and in which at present our therapeutic resources are extremely limited. I shall refer to two of these which are in a sense characteristic of early life—that is to say, they are much more common in childhood and youth than in later years. These two types are acute general carditis and adherent pericardium. There is a third type which is easily overlooked in adoles-

cence, but which it is important to bear in mind, viz. acute ulcerative endocarditis.

The form which I have described as **acute general carditis** is frequently a rapidly fatal form of disease. It is the result as a rule of a fresh rheumatic infection attacking a previously damaged heart, although it may occur without any history or evidence of pre-existing disease. It is often described as acute pericarditis, because the signs of that condition are well marked, but this is merely a part and not necessarily the most important part of the carditis. It will usually be found that pericardium, endocardium, and myocardium share in the acute inflammatory process, and of these the most important is the myocardium. Probably the fatal issue which so often follows depends chiefly on the failure of the heart muscle, the result of myocarditis.

The clinical picture is a very striking one. The signs of illness develop rapidly after a few days of malaise, feverishness, shortness of breath, and extreme lassitude. The patient becomes excited, restless, and irritable. Pain may be complained of in the præcordial or shoulder regions, and is often severe. The breathing becomes more rapid and more laboured and orthopnœa soon becomes marked. Paroxysms of dyspnœa like cardiac asthma occur. Sleep is irregular and broken by starts or attacks of breathlessness, vomiting is often present, and the liver is much enlarged. The veins of the neck are often prominent and pulsating strongly. A tendency to cyanosis about the ears and extremities is in

marked contrast to the progressive pallor about the face. Œdema of the limbs is not usually present except as a terminal phenomenon. We have, in short, all the signals pointing to a heart in distress.

When the heart is examined there is no lack of confirmatory signs. It is probably pulsating over a large area and dilated in all its dimensions. The action is rapid and feeble, and as a rule is regular, as judged by the apex beat. Pericardial friction is very often present, but may disappear on the occurrence of fluid exudation. Murmurs are not wanting, chiefly associated with the mitral valve, where a systolic and diastolic murmur may often be heard, varying in character from time to time. Tricuspid and aortic murmurs may also be present. The large area of cardiac pulsation suggests overaction of the heart, and that it is not efficient action is shown by the state of the pulse, which is usually feeble, dierotic, often collapsing, and always badly sustained.

Such an attack may be recovered from by the subsidence of the acute inflammation or the absence of further complications. The post-mortem appearances in fatal cases go far to explain the clinical phenomena. Signs of pericarditis and endocarditis may be present to a varying extent but are usually well marked. Dilatation of all the cavities and of the valvular openings will usually be present. In and about the heart there are well-marked signs of inflammatory exudation, and in severe cases this may be hæmorrhagic in character. The myocardium

is soft, swollen, congested, or hæmorrhagic. On microscopic examination it presents the signs of acute inflammation, with exudation of cells, fatty degeneration of the fibres, and possibly hæmorrhagic areas. The following may be taken as a typical example of this form of fulminating carditis :

Case XIII. A female child of six years was admitted to hospital with a history of two days' illness, characterized by pain about the heart and left shoulder, shortness of breath, loss of appetite, sleeplessness, and depression. There was no history of rheumatic fever, but eight months previously the mother was told by a doctor that the child had heart disease, which caused the shortness of breath from which she suffered. Six months previously the child had been in hospital with bronchitis. At that time the heart was found to be slightly dilated, and there was a systolic murmur following the first sound at the apex and conducted well into the axilla. The bronchitic signs soon disappeared, but on the fourth and sixth days after admission she had attacks of severe dyspnœa, lasting for a short time, and regarded as 'asthmatic' in character. She made a good recovery.

On admission the temperature was 102°, the pulse 140, and the respiration 72. The child looked very ill, the face was pale, and the expression anxious and pained. A troublesome cough was present which aggravated the pain about the heart. There was orthopnœa, and the lungs presented the signs of bronchitis and œdema. There was diffused præ-

cordial pulsation, and both sides of the heart were dilated. A well-marked pericardial rub was present over an area extending from the apex to the sternum. Intra-cardiac murmurs were difficult to distinguish, but a systolic murmur could be heard at the apex. The liver was much enlarged, and the veins of the neck pulsated visibly.

The illness ran an irregular course, but there was never any definite improvement. The temperature fluctuated, but seldom rose above 100·5° F. The area of cardiac dullness increased, extending one and a half inches beyond the nipple line on the left, and three-quarters of an inch beyond the right border of the sternum. This was clearly due to dilatation of the heart, as pericardial friction was also present and extending. Pleural friction appeared in the right and then in the left axillary region. Pain in the left shoulder was much complained of. This seemed to be definitely cardiac in origin, for there was no evidence of arthritis and the arm could be moved freely without discomfort. There was always a mitral systolic murmur present and sometimes a diastolic murmur. The pulse continued rapid, averaging about 120, and was soft and dicrotic. Coughing was often troublesome. Dyspnœa was always present, and was sometimes aggravated by paroxysmal attacks of great severity. Vomiting occurred at intervals. Towards the end—which came on the eighteenth day of the illness—some œdema of the lower extremities and the vulva was present. The last of the notes made by my house-physician, Dr. Hay, states that after a hypo-

dermic injection of morphia and digitalin she had a better night, and was comfortable and drowsy in the morning. There was little change in the physical signs, but she was passing little urine and the œdema of the feet was increasing. She had a fairly quiet day, sleeping a good deal, but always looking extremely ill. The pulse continued unchanged at 120 per minute until midnight, when it suddenly failed and became imperceptible. The child became very restless for half an hour and wanted to get on to a chair. She then slept off and on for five hours, passing away quietly in her sleep at the end of that time, during which the pulse at the wrist had been imperceptible.

At the autopsy the pericardium was found to be adherent to the heart, intimately so at the base. The pericardium was thickened, acutely congested, and lined with gelatinous exudate. The heart was very much enlarged. The walls were congested and on section dripped blood. All the cavities were dilated. The valves presented no evidence of recent change, but the border of the mitral valve was thickened and puckered. There was collapse of the lower lobe of the left lung, and a patch of pleural thickening and congestion (recent) over the base of the right lung.

We seem to have in the post-mortem findings in such a case a sufficient explanation of the acute cardiac distress and marked cardiac signs present during life. Yet a doubt arose in my mind as to whether in such a case the strain thrown on the

heart was always in itself sufficient to explain the fatal issue. The youthful heart is capable of much, and even from very severe attacks of general carditis one has seen recovery follow. In the case described above careful polygraphic tracings were taken, and from them I was led to believe that in addition to the signs of cardiac disease noted there was also a disordered rhythm of the heart. The tracings

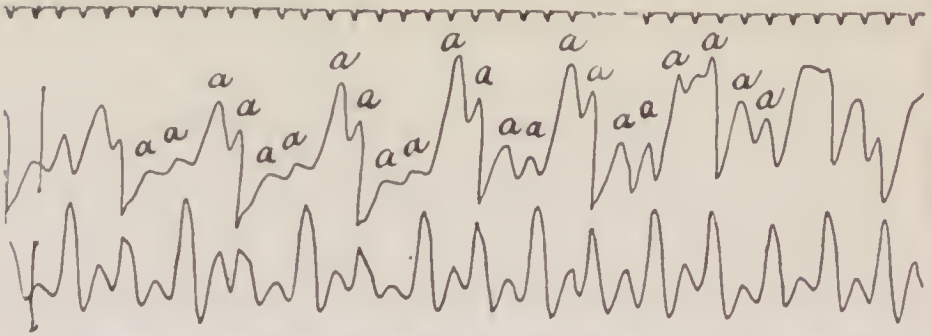


Fig. 49. Auricular flutter in acute carditis. Jugulo-brachial tracing. The venous waves occur at the rate of 250 per minute, and it is suggested that each is due to an auricular contraction. The ventricles are contracting at one-half that rate, namely, 125 times per minute (A : V :: 2 : 1).

would seem to show that while the ventricle was beating at 125 per minute, the auricle was beating at twice that rate, namely 250 (Fig. 49); and a few days later, while the ventricular rate was 130 the auricular rate was three times as great, namely 390 per minute (Fig. 50). Such a disturbance of the normal rhythm would necessarily upset the mechanism of the heart, i.e. the orderly sequence in which each auricular contraction is followed by a ventricular one. It would further tend to affect seriously the action of the left

ventricle, as happens in chronic cases of heart disease when such a disturbance is present. The condition was apparently one of auricular flutter, in which some new focus of stimulation arose in the auricle and dominated the rate of auricular contraction. Instead of responding to each of these auricular contractions the ventricle responded only to every second or third one. In support of this view there is further evidence

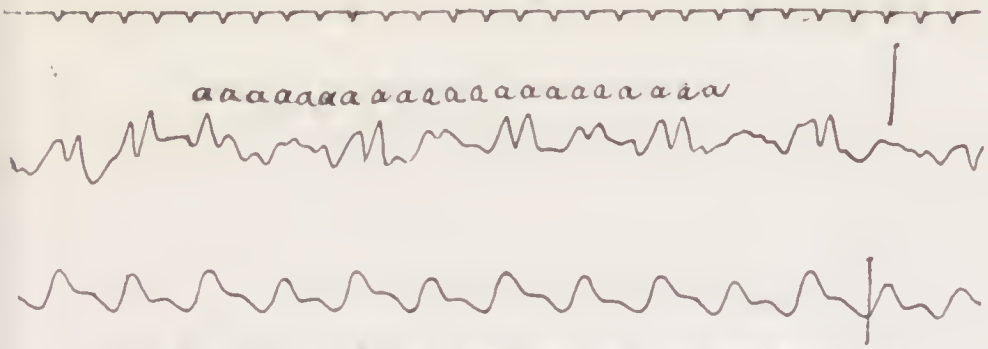


FIG. 50. Auricular flutter in acute carditis. Same case seven days later. Jugulo-brachial tracing. The venous waves here number 390 per minute, and it is suggested that each is due to an auricular contraction. The ventricles are contracting at one-third of that rate, namely, 130 times per minute (A : V :: 3 : 1). The *pulsus alternans* is present in the brachial tracing.

in the shape of the *pulsus alternans*, as shown in the tracings, and Dr. Mackenzie informs me that that form of pulse is found in the young only with some such abnormal rhythm.

Further investigations will be required to determine whether in these cases of acute general carditis a disordered rhythm is often present. If it should be found I should be more hopeful of the treatment, for a disordered rhythm will respond to treatment

by digitalis much better than a ventricle weakened by acute inflammation will. If we can control the rhythm so as to relieve the left ventricle, we shall place the patient in a much better position for weathering the attack. On the other hand, if we cannot control the rhythm it may be found that the prognosis is made materially worse by the development of this abnormal auricular action as compared with those cases in which no such disorder of the mechanism is present.

We do not obtain any benefit in these cases of acute general carditis by means of salicylate of soda. In fact, when there are signs of acute cardiac failure I think the drug often does harm, and certainly increases the tendency to vomiting. Digitalis is of no use when used as a means of increasing the contractile power of the heart in cases of acute inflammation. As a means of checking a disturbed rhythm through its action on the vagus it may prove beneficial, but of this I have no experience. We have, of course, to secure as complete physical rest to the patient as possible. This is best obtained by giving opium or morphia freely. The general strength will be maintained up to a certain point by hypodermic injections of strychnine and by moderate doses of brandy. As a rule, the relief of symptoms is the most we can do in such cases.

Another of the serious forms of organic heart disease often associated with cardiac failure is that known as **adherent pericardium**. Many forms of this may be present without affecting the cardiac

action in any way, in which case they are probably unrecognized. The clinical forms are those in which the functional activity of the heart is so interfered with that compensatory hypertrophy is present. If the myocardium is extensively diseased, such hypertrophy is scarcely possible, so that the presence of hypertrophy presupposes the existence of a good deal of healthy muscle. The situation of the adhesions will determine the situation of the most marked hypertrophy, i.e. whether of the right heart or of the left, but in most cases there will be a general enlargement of the heart. If one recognizes that in this condition the cardiac trouble is a mechanical one, due to causes outside the heart, it is difficult to see how it can be benefited by any medicinal treatment. The heart is already doing its best, as shown by the exaggerated action which leads to a bulging forward of the whole chest wall in the præcordial region. With all this heaving action of the heart the pulse is often small or collapsing, showing that the ventricle is never completely emptied, but in spite of its size is only barely competent to carry on the circulation.

Case XIV. Female, thirteen years. At the age of five years this girl had an attack of rheumatic fever, but her health continued good until the age of eleven years. She had taken part in school life and school games without the consciousness of any disability. An attack of subacute rheumatism took place when she was eleven, with pains in the joints, and the heart was found to be affected. Symptoms

of cardiac failure developed and were only partially recovered from. For the fourteen months before admission to hospital she had been confined to bed.

She was a poorly developed girl and looked younger than her years. There were numerous signs of cardiac failure, namely, œdema of the lungs, much enlarged liver, cyanosis, and slight œdema of the extremities. The breathing was laboured, orthopnœa was present, coughing disturbed the patient, and præcordial pain was complained of. The præcordial region was bulged forwards, and a marked heaving impulse was visible and palpable. The cardiac dullness was much enlarged, extending on the left two and a half inches outside the nipple line and one and a half inches to the right to the sternum. There was a loud musical systolic murmur in the mitral area, audible also over a wide area of the chest, back and front. There was no evidence of other valvular involvement.

Under rest in bed the acute symptoms were relieved, but the orthopnœa persisted and the liver did not diminish in size. The pulse was regular but rapid (120), and nothing seemed to have any effect in reducing the rate. Tracings showed that it was dicrotic in character, and it felt collapsing to the finger. Digitalis was tried for three weeks in full doses, namely, half a dram of the tincture daily, but without any effect on the pulse rate or otherwise.

After two months' rest in bed the patient was allowed to get up and rest in a chair. This was followed by œdema of the lower extremities and no

improvement. She began to suffer from præcordial pain, chiefly over the apex region, which necessitated the use of morphia; the signs of cardiac failure increased, and œdema of the lower extremities and abdomen became more pronounced. Death occurred quite suddenly three months after admission.

At the necropsy the heart was found to be greatly enlarged in all its dimensions. The heart was firmly adherent to the chest wall at the apex. The two layers of the pericardium were adherent and greatly thickened throughout their whole extent. All the cavities of the heart were much dilated and their walls thinned. The mitral valve admitted three fingers and showed a few small vegetations along its margin. Similar vegetations were present on the aortic valve. The liver showed a typical nutmeg appearance, and both it and the spleen were much enlarged.

Although in cases of adherent pericardium there may be some myocardial and endocardial trouble, the important lesion is the tying down of the heart and the limitation of its action by adhesion to the chest wall or surrounding structures. The result is a gradual wearing down of the cardiac powers. The end may be hastened by subacute attacks of rheumatic carditis. Under rest and good surroundings it is sometimes surprising how long these patients with enormous hearts may survive.

In several cases of adherent pericardium I have tested digitalis thoroughly and have found no benefit. When there are no symptoms of cardiac failure the

best treatment is to regulate the amount of work thrown on the heart so that breathlessness and exhaustion are not induced. When symptoms of cardiac failure appear the amount of exertion must be further curtailed and a completely sedentary life is often necessary. When œdema begins, one can try digitalis, but my own experience has been that such a symptom is usually the prelude to complete heart failure.

The question of operation has been raised within recent years. Such an operation as the removal of adhesions may be regarded as theoretically possible but not usually practicable. In one case, with great bulging of the præcordia, Mr. Clayton-Greene divided the costal cartilages of the 4th, 5th, 6th, and 7th ribs on the left side, with the view of giving the heart more space. When divided the cartilages sprang apart for some distance. The patient was a boy of twelve and he lived for a year afterwards. It appeared to me that he was to some extent relieved and was able to make more exertion without discomfort after the operation than before. Probably this is as much as one can expect from the operation considering the gravity of the underlying heart disease.

The last form of heart disease to be considered is the so-called **ulcerative** or **infective** or **malignant endocarditis**. Although there is usually a pathological basis in proliferative ulceration of a valve, and ulceration extending along the chambers of the heart or blood-vessels, the disease is really a form

of septicæmia, and is to be treated as such. It may be regarded as a complication of rheumatic carditis, which in the great majority of cases leads to a fatal termination. Out of sixteen cases below the age of twelve years observed by Poynton, in seven there was a previous history of rheumatic fever, four were pneumococcal, two were staphylococcal, and three were of doubtful origin. The course of the illness may be insidious and prolonged, and it may for some time be difficult to distinguish it from the condition I have referred to as progressive carditis. The distinction is an important one because treatment in the latter case is much more hopeful. As distinguishing features in infective endocarditis one may point to the irregular hectic temperature, the inefficacy of salicylate treatment in reducing the temperature or altering the symptoms, and the evidences of systemic infection which usually develop.

Case XV. A boy of thirteen years had an attack of acute rheumatism, with pain and swelling in the knees and knuckles. When he came under observation a month later he looked extremely ill, very pale and tired. On physical examination there were no such objective findings about the heart as his appearance had suggested. The apex beat was just outside the nipple line, and a systolic murmur of an organic character was present at that spot and was conducted well into the axilla. The cardiac rate was persistently rapid, 112 to 120 per minute. He was very breathless on any exertion.

During the first three weeks in hospital the temperature rose to 100° F. occasionally at night, but as a rule was not over 99°. The cardiac rate continued rapid. A diastolic murmur developed at the base of the heart, and was audible down the left side of the sternum. It was clearly aortic in origin, and its presence was accompanied by a change in the character of the pulse, which became collapsing. The blood-pressure was 120 mm. There were no active signs of rheumatism, and the boy's general appearance was much improved.

During the second three weeks the temperature began to rise more constantly to 100° F. at night. The pulse continued rapid, dicrotic, and collapsing. There were no signs of right-sided failure, but the apex beat gradually extended outwards to one inch beyond the nipple line. The boy's general condition was satisfactory. A slight attack of dry pleurisy on the right side caused trouble for a few days.

During the third three weeks the temperature became definitely hectic in type, varying from 97° in the morning to 101° at night. Following an attack of pleurisy on the left side, consolidation of the left lung appeared at the base posteriorly and spread gradually up to the apex. Præcordial pain and tenderness were marked. The cardiac dilatation increased. A staphylococcus was grown in pure culture from the patient's blood. Progressive anæmia and weakness led to a fatal termination.

Treatment of this affection is to be directed to the maintenance of the patient's strength as far

as possible and to the relief of symptoms. An organism can often be detected in the blood, and I have employed autogenous vaccines in several cases, but without any definite results in the way of cure or even amelioration. Nor have polyvalent sera proved of any service in my hands. If one views the condition as one of general septicæmia, and if one considers the extensive cardiac lesions usually found post-mortem, it seems clear that our present therapeutic resources are not likely to cope successfully with the disease.

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